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PREHOSPITAL MANAGEMENT OF THE INJURED ANIMAL

SURVEY OF THE SCENE

1. CALL FOR HELP! At the accident scene, it usually takes more than one person to assist the animal and prevent injury to the animal and human bystanders.
2. If an accident has occurred in a traffic zone, alert oncoming traffic of the injured animal in the road. Make sure you have a piece of clothing or other object to alert oncoming traffic. Do not become injured yourself because oncoming traffic cannot see or identify you.
3. If the animal is conscious, prevent yourself from becoming injured while moving the animal to a safe location. Use a belt, rope, or piece of long cloth to make a muzzle to secure around the animal's mouth and head. If this is not possible, cover the animal's head with a towel, blanket, or coat before moving it.
4. If the animal is unconscious or is unconscious and immobile, move it to a safe location with a back support device that can be made from a box, door, flat board, blanket, or sheet.

INITIAL EXAMINATION

1. Is there a patent airway? If airway noises are present or the animal is stuporous, gently and carefully extend the head and neck. If possible, extend the tongue. Wipe mucus, blood, or vomitus from the mouth. In unconscious animals, maintain head and neck stability.
2. Look for signs of breathing. If there is no evidence of breathing or the gum color is blue, begin mouth-to-nose breathing. Encircle the muzzle area with your hands to pinch down on the gums, and blow into the nose 15 to 20 times per minute.
3. Is there evidence of cardiac function? Check for a palpable pulse on the hind legs or for an apex beat over the sternum. If no signs of cardiac function are found, begin external cardiac compressions at 80 to 120 times per minute.
4. Is there any hemorrhage? Use a clean cloth, towel, paper towel, or disposable diaper or feminine hygiene product to cover the wound. Apply firm pressure to slow hemorrhage and prevent further blood loss. Do not use a tourniquet because this can cause further damage. Apply pressure, and as blood seeps through the first layer of bandage material, place a second layer over the top.
5. Cover any external wounds. Use a clean bandage material soaked in warm water, and transport the animal to the nearest veterinary emergency facility. Address penetrating wounds to the abdomen and thorax immediately.
6. Are there any obvious fractures present? Immobilize fractures with homemade splints made of newspaper, broom handles, or sticks. Muzzle the awake animal before

attempting to place any splints. If a splint cannot be attached safely, place the animal on a towel or blanket and transport the animal to the nearest veterinary emergency facility.

7. Are there any burns? Place wet, cool towels over the burned area and remove as the compress warms to body temperature.
8. Wrap the patient to conserve heat. If the animal is shivering or in shock, wrap it in a blanket, towel, or coat and transport it to the nearest veterinary emergency facility.
9. Is the animal suffering from heat-induced illness (heat stroke)? Cool the animal with room-temperature wet towels (NOT COLD) and transport it to the nearest veterinary emergency facility.

PREPARATION FOR TRANSPORT

1. Call ahead! Let the facility know that you are coming. Be prepared by having emergency numbers and locations available. The police or sheriff's department may be able to aid in locating the nearest veterinary emergency facility.
2. Line upholstery with plastic bags or sheeting to prevent soilage, when possible.
3. Move the injured patient carefully. Use the same approach as moving the animal from the pavement.
4. DRIVE SAFELY. Do not turn one accident into two. Ideally, have a bystander or friend or family member drive while another person stays in the backseat with the animal.

INITIAL EMERGENCY EXAMINATION, MANAGEMENT, AND TRIAGE

Examination of the acutely injured animal that is unconscious, in shock, or suffering from acute hemorrhage or respiratory distress must proceed simultaneously with immediate aggressive lifesaving treatment. Because there often is no time for detailed history taking, diagnosis is largely based on the physical examination findings and simple diagnostic tests. Triage is the art and practice of being able to assess patients rapidly and sort them according to the urgency of treatment required. Immediate recognition and prompt treatment potentially can be lifesaving.

PRIMARY SURVEY AND EMERGENCY RESUSCITATION MEASURES

Perform a brief but thorough systematic examination of the animal, noting the most important ABCs of any emergent patient.

A = AIRWAY

Is the airway patent? Pull the patient's tongue forward and remove any debris obstructing the airway. Suction and a laryngoscope may be necessary. If necessary, intubate or place a transtracheal oxygen source. An emergency tracheostomy may be necessary if upper airway obstruction is present and cannot be resolved immediately with the foregoing measures.

B = BREATHING

Is the animal breathing? If the animal is not breathing, immediately intubate the animal and start artificial ventilations with a supplemental oxygen source (see Cardiac Arrest and Cardiopulmonary Cerebral Resuscitation).

If the animal is breathing, what is the respiratory rate and pattern? Is the respiratory rate normal, increased, or decreased? Is the respiratory pattern normal, or is the breathing rapid and shallow, or slow and deep with inspiratory distress? Are the respiratory noises normal, or is there a high-pitched stridor on inspiration characteristic of an upper airway obstruction? Does the animal have its head extended and elbows abducted away from the body

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with orthopnea? Do the commissures of the mouth move with inhalation and exhalation? Is there evidence of expiratory distress with an abdominal push upon exhalation? Note the lateral chest wall. Do the ribs move out and in with inhalation and exhalation, or is there paradoxical chest wall motion in an area that moves in during inhalation and out during exhalation, suggestive of a flail chest? Is there any subcutaneous emphysema that suggests airway injury?

Auscultate the thorax *bilaterally*. Are the breath sounds normal? Do they sound harsh with crackles because of pneumonia, pulmonary edema, or pulmonary contusions? Are the lung sounds muffled because of pleural effusion or pneumothorax? Are there inspiratory wheezes in a cat with bronchitis (asthma)? What is the mucous membrane color? Is it pink and normal, or is it pale or cyanotic? Palpate the neck, lateral thorax and dorsal cervical region to check for tracheal displacement, subcutaneous emphysema, or rib fractures.

C = CIRCULATION

What is the circulatory status? What is the patient's heart rate and rhythm? Can you hear the heart, or is it muffled because of hypovolemia, pleural or pericardial effusion, pneumothorax, or diaphragmatic hernia? Palpate the pulses. Is the pulse quality strong and regular and synchronous with each heartbeat, or are there thready, dropped pulses? What are the patient's electrocardiogram (ECG) rhythm and blood pressure?

Is there arterial hemorrhage? Note whether there is any bleeding present. Use caution if there is any blood on the fur. Wear gloves. The blood may be from the patient, and gloves will help prevent further contamination of any wounds; or the blood may be from a good Samaritan bystander. If external wounds are present, note their character and condition. Place a pressure bandage on any arterial bleeding or external wounds to prevent further hemorrhage or contamination with nosocomial organisms.

Establish large-bore vascular or intraosseous access (See VASCULAR ACCESS TECHNIQUES). If hypovolemic or hemorrhagic shock is present, institute immediate fluid resuscitation measures. Start with one fourth of a calculated shock dose of crystalloid fluids ($0.25 \times [90 \text{ mL/kg/hour for dogs}]$; $0.25 \times [44 \text{ mL/kg/hour for cats}]$), and reassess perfusion parameters of heart rate, capillary refill time, and blood pressure. If pulmonary contusions are suspected, use of a colloid such as hetastarch at 5 mL/kg in incremental boluses can improve perfusion with a smaller volume of fluid. In cases of head trauma, hypertonic (7%) sodium chloride (saline) can be administered (4 mL/kg IV bolus) with hetastarch or Dextran-70 (10 mL/kg). Acute abdominal hemorrhage caused by trauma can be tamponaded with an abdominal compression bandage.

After the immediate ABCs, proceed then with the rest of the physical examination and treatment by using the mnemonic A CRASH PLAN.

A = AIRWAY

C AND R = CARDIOVASCULAR AND RESPIRATORY

A = ABDOMEN

Palpate the patient's abdomen. Is there any pain or are there any penetrating injuries present? Look at the patient's umbilicus. Reddening around the umbilicus can suggest intraabdominal hemorrhage. Is there a fluid wave or mass palpable? Examine the inguinal, caudal, thoracic, and paralumbar regions. Clip the fur to examine the patient for bruising or penetrating wounds. Percuss and auscultate the abdomen for borborygmi.

S = SPINE

Palpate the animal's spine for symmetry. Is there any pain or obvious swelling or fracture present? Perform a neurologic examination from C1 to the last caudal vertebra.

H = HEAD

Examine the eyes, ears, mouth, teeth, nose, and all cranial nerves. Stain the eyes with fluorescence dye to examine for corneal ulcers in any case of head trauma. Is there anisocoria or Horner syndrome present?

P = PELVIS

Perform a rectal examination. Palpate for fractures or hemorrhage. Examine the perineal and rectal areas. Examine the external genitalia.

L = LIMBS

Examine the pectoral and pelvic extremities. Are there any obvious open or closed fractures? Quickly splint the limbs to prevent further damage and help control pain. Examine the skin, muscles, and tendons.

A = ARTERIES

Palpate the peripheral arteries for pulses. Use a Doppler piezoelectric crystal to aid in finding a pulse if thromboembolic disease is present. Measure the patient's blood pressure.

N = NERVES

From afar, note the level of consciousness, behavior, and posture. Note respiratory rate, pattern, and effort. Is the patient conscious, or is the patient obtunded or comatose? Are the pupils symmetric and responsive to light, or is there anisocoria present? Does the patient display any abnormal postures such as Schiff-Sherington (extended rigid forelimbs, flaccid paralysis of the hind limbs) that may signify severe spinal shock or a severed spinal cord? Examine the peripheral nerves for motor and sensory input and output to the limbs and tail.

ANCILLARY DIAGNOSTIC EVALUATION

Hemodynamic techniques: Perform electrocardiography, direct or indirect blood pressure monitoring, and pulse oximetry in any critically ill traumatized patient.

Imaging techniques: Obtain radiographs of the thorax and abdomen in any animal that has sustained a traumatic injury once the patient is more stable and can tolerate positioning for the procedures. Survey radiographs may reveal pneumothorax, pulmonary contusions, diaphragmatic hernia, pleural or abdominal effusion, or pneumoperitoneum.

Laboratory testing: Immediate diagnostic testing should include a hematocrit, total solids, glucose, blood urea nitrogen (BUN)/azostick, and urine specific gravity. Ancillary diagnostic tests that can be performed soon thereafter include a complete blood count and peripheral blood smear to evaluate platelet count and red and white blood cell morphology. Also consider arterial blood gas and electrolytes, coagulation parameters (activated clotting time [ACT], prothrombin time [PT], activated partial thromboplastin time [APTT]), serum biochemistry profile, serum lactate, and urinalysis.

Invasive testing: Invasive diagnostic techniques that may need to be performed include thoracocentesis, abdominal paracentesis, and diagnostic peritoneal lavage.

SUMMARY OF PATIENT STATUS

After completing the initial physical examination, answer the following questions: What supportive care is required at this time? Are additional diagnostic procedures needed? If so, which procedures, and is the patient stable enough to tolerate those procedures without further stress? Should an additional period of observation be instituted before further definitive treatment plans are undertaken? Is immediate surgical intervention necessary? Is additional supportive care required before surgery? What anesthetic risks are evident?

THE RAPIDLY DECOMPENSATING PATIENT

Animals that do not respond to initial resuscitation usually have severe ongoing or preexisting physiologic disturbances that contribute to severe cardiovascular and metabolic instability. A patient that does not respond to or responds to and then stops responding to

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BOX 1-1 CLINICAL SIGNS OF DECOMPENSATION

Weak or poor peripheral pulse quality
 Cool peripheral extremities
 Cyanosis or muddy-colored (gray) mucous membranes
 Pale mucous membranes
 Prolonged capillary refill time
 Increased or decreased body temperature
 Decreased renal output in a euvolemic patient
 Inappropriate mentation or confusion
 Depression
 Tachycardia or bradycardia
 Declining hematocrit
 Distended, painful abdomen
 Cardiac dysrhythmia
 Abnormal respiratory pattern
 Respiratory difficulty or distress
 Gastrointestinal blood loss via hematemesis or in feces

BOX 1-2 CAUSES OF ACUTE DECOMPENSATION

Acute renal failure
 Acute respiratory distress syndrome
 Bowel and gastric rupture
 Cardiac dysrhythmia
 Central nervous system edema and hemorrhage, and brainstem herniation
 Coagulopathies including disseminated intravascular coagulation
 Internal hemorrhage
 Multiple organ dysfunction syndrome
 Pneumothorax
 Pulmonary contusions
 Pulmonary thromboembolism
 Sepsis or septic shock
 Systemic inflammatory response syndrome
 Urinary bladder rupture

initial resuscitation efforts should alert the clinician that decompensation is occurring (Boxes 1-1 and 1-2).

Additional Reading

- Ettinger SJ, Feldman EC, editors: *Critical care*. In *Textbook of veterinary internal medicine*, ed 6, St Louis, 2005, Elsevier-Saunders.
- Mathews KA: *Veterinary emergency and critical care manual*, Guelph, Ontario, Canada, 1996, Lifelearn.
- Wingfield WE: Decision making in veterinary emergency medicine. In Wingfield WE, editor: *Veterinary emergency secrets*, ed 2, Philadelphia, 2001, Hanley & Belfus.
- Wingfield WE: Treatment priorities in trauma. In Wingfield WE, editor: *Veterinary emergency secrets*, ed 2, Philadelphia, 2001, Hanley & Belfus.

EMERGENCY DIAGNOSTIC AND THERAPEUTIC PROCEDURES

ABDOMINAL PARACENTESIS AND DIAGNOSTIC PERITONEAL LAVAGE

Abdominocentesis (abdominal paracentesis) refers to puncture into the peritoneal cavity for the purpose of fluid collection. Abdominal paracentesis is a sensitive technique for fluid collection as long as more than 6 mL/kg of fluid is present within the abdominal cavity.

In the event that you suspect peritonitis and have a negative tap with abdominal paracentesis, a diagnostic peritoneal lavage can be performed.

To perform abdominal paracentesis, follow this procedure:

1. Place the patient in left lateral recumbency and clip a 4- to 6-inch square with the umbilicus in the center.
2. Aseptically scrub the clipped area with antimicrobial scrub solution.
3. Wearing gloves, insert a 22- or 20-gauge needle or over-the-needle catheter in four quadrants: cranial and to the right, cranial and to the left, caudal and to the right, and caudal and to the left of the umbilicus. As you insert the needle or catheter, gently twist the needle to push any abdominal organs away from the tip of the needle. Local anesthesia typically is not required for this procedure, although a light sedative or analgesic may be necessary if severe abdominal pain is present. In some cases, fluid will flow freely from one or more of the needles. If not, gently aspirate with a 3- to 6-mL syringe or aspirate with the patient in a standing position. Avoid changing positions with needles in place because iatrogenic puncture of intraabdominal organs may occur.
4. Save any fluid collected in sterile red- and lavender-topped tubes for cytologic and biochemical analyses and bacterial culture. Monitor hemorrhagic fluid carefully for the presence of clots. Normally, hemorrhagic effusions rapidly become defibrinated and do not clot. Clot formation can occur in the presence of ongoing active hemorrhage or may be due to the iatrogenic puncture of organs such as the spleen or liver.

If abdominal paracentesis is negative, a diagnostic peritoneal lavage can be performed. Peritoneal dialysis kits are commercially available but are fairly expensive and often impractical.

To perform a diagnostic peritoneal lavage, follow this procedure:

1. Clip and aseptically scrub the ventral abdomen as described previously.
2. Wearing sterile gloves, cut multiple side ports in a 16- or 18-gauge over-the-needle catheter. Use care to not cut more than 50% of the circumference of the catheter, or else the catheter will become weakened and potentially can break off in the patient's abdomen.
3. Insert the catheter into the peritoneal cavity caudal and to the right of the umbilicus, directing the catheter dorsally and caudally.
4. Infuse 10 to 20 mL of sterile lactated Ringer's solution or 0.9% saline solution that has been warmed to the patient's body temperature. During the instillation of fluid into the peritoneal cavity, watch closely for signs of respiratory distress because an increase in intraabdominal pressure can impair diaphragmatic excursions and respiratory function.
5. Remove the catheter.
6. In ambulatory patients, walk the patient around while massaging the abdomen to distribute the fluid throughout the abdominal cavity. In nonambulatory patients, gently roll the patient from side to side.
7. Next, aseptically scrub the patient's ventral abdomen again, and perform an abdominal paracentesis as described previously. Save collected fluid for culture and cytologic analyses; however, biochemical analyses may be artifactually decreased because of dilution. Remember that you likely will retrieve only a small portion of the fluid that you instilled.

Additional Reading

- Bjorling DE, Latimer KA, Rawlings CA, et al: Diagnostic peritoneal lavage before and after abdominal surgery in dogs, *Am J Vet Res* 44(5):816-820, 1983.
- Crowe DT: Abdominocentesis and diagnostic peritoneal lavage in small animals, *Mod Vet Pract* 13:877-882, 1984.
- Crowe DT: Diagnostic abdominal paracentesis techniques: clinical evaluation in 129 dogs and cats, *J Am Anim Hosp Assoc* 20:223-230, 1984.
- Walters JM: Abdominal paracentesis and diagnostic peritoneal lavage, *Clin Tech Small Anim Pract* 18(1):32-38, 2003.

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BANDAGING AND SPLINTING TECHNIQUES

In general bandages can be applied to open or closed wounds. Bandaging is used for six general wound types: open contaminated or infected wounds, open wound in the repair stage of healing, a closed wound, a wound in need of a pressure bandage, a wound in need of pressure relief, and a wound in need of immobilization. Box 1-3 lists various functions of bandages.

The materials and methods of bandaging depend on the type of injury, the need for pressure and immobilization, the need to prevent pressure, and the stage of healing. In general, bandage material has three component layers. If pressure relief or immobilization is required, splint material also may be incorporated into the bandage. The contact layer is the layer of bandage material that actually is adjacent to the wound itself. The secondary or intermediary layer is placed over the contact (primary) layer. Finally, the outer tertiary layer covers the bandage and is exposed to the outside.

OPEN CONTAMINATED AND INFECTED WOUNDS

Open contaminated or infected wounds often have large amounts of necrotic tissue and foreign debris and emit copious quantities of exudate. The contact layer used in an open contaminated or infected wound should be wide-mesh gauze sponges with no cotton filling. The sponges can be left dry if the wound has minimal exudate but should be moistened with sterile 0.9% saline or lactated Ringer's solution if the wound has high-viscosity exudate. Topical ointments may be applied (silver sulfadiazine, chlorhexidine ointment) if necessary. The intermediate layer should be thick absorbent wrapping material, covered by an outer layer of porous tape: Elastikon (Johnson & Johnson Medical, Arlington, Texas), or Vetrap (3M, St. Paul, Minnesota). Change the bandages at least once daily or more frequently if strike-through of exudate occurs through the bandage.

To place a wet-to-dry bandage over a wound, first place the contact layer over the wound. Next, place strips of adhesive tape to the patient's paw on either side, if possible. The strips (stirrups) will be used to hold the bandage in place and prevent it from slipping down the limb. Wrap the intermediate layer over the contact layer. Turn the adhesive strips around so that the adhesive layer can be secured to the intermediary layer in place. Wrap the final, or tertiary layer over the bandage.

The function of a wet-to-dry bandage is to help debride a wound. The moistened gauze dries and is pulled off the wound at each bandage change. Dry necrotic tissue and debris that is adhered to the gauze is pulled off with it. In addition, the moistened material dilutes the wound exudates and enhances its absorption into the gauze contact layer. If large amounts of exudate come from the wound, the contact layer and intermediate layer absorb the exudate, wicking the material away from the wound. Finally, delivery of medications into the wound can occur to promote the development of healthy granulation tissue.

BOX 1-3 FUNCTIONS OF BANDAGES AND SPLINTS

Exert pressure	Protect a wound from environmental bacteria
Obliterate dead space	Protect the environment from wound blood, exudate, and bacteria
Reduce edema	Immobilize a wound and support underlying osseous structures
Minimize hemorrhage	Minimize patient discomfort
Prevent pressure on wounds	Serve as a vehicle for antiseptics and antibiotics
Decubitus ulcers	Serve as an indicator of wound secretions
Pack a wound	Provide an aesthetic appearance
Wet-to-dry bandages for deep shearing injuries	
Absorb exudate and debride a wound	
Wet-to-dry bandages	

OPEN WOUND IN REPAIR STAGE OF HEALING**Early repair**

During the early stage of repair, granulation tissue, some exudate, and minor epithelialization is observed. Place a nonadherent bandage with some antibacterial properties (petroleum or nitrofurazone-impregnated gauze) or absorbent material (foam sponge, hydrogel, or hydrocolloid dressing) in direct contact with the wound to minimize disruption of the granulation tissue bed. Next, place an absorbent intermediate layer, followed by a porous outer layer, as previously described. Granulation tissue can grow through gauze mesh or adhere to foam sponges and can be ripped away at the time of bandage removal. Hemorrhage and disruption of the granulation tissue bed can occur.

Late repair

Later in the repair process, granulation tissue can exude sanguineous drainage and have some epithelialization. A late nonadherent bandage is required. The contact layer should be some form of nonadherent dressing, foam sponge, hydrogel, or hydrocolloid substance. The intermediate layer and outer layers should be absorbent material and porous tape, respectively. With nonadherent dressings, wounds with viscous exudates may not be absorbed well. This may be advantageous and enhance epithelialization, provided that complications do not occur. Infection, exuberant granulation tissue, or adherence of absorbent materials to the wound may occur and delay the healing process.

MOIST HEALING

Moist healing is a newer concept of wound management in which wound exudates are allowed to stay in contact with the wound. In the absence of infection a moist wound heals faster and has enzymatic activity as a result of macrophage and polymorphonuclear cell breakdown. Enzymatic degradation or “autolytic debridement” of the wound occurs. Moist wounds tend to promote neutrophil and macrophage chemotaxis and bacterial phagocytosis better than use of wet-to-dry bandages. A potential complication and disadvantage of moist healing, however, is the development of bacterial colonization, folliculitis, and trauma to wound edges that can occur because of the continuously moist environment.

Use surfactant-type solutions (Constant Clens; Kendall, Mansfield, Massachusetts) for initial wound cleansing and debridement. Use occlusive dressings for rapid enzymatic debridement with bactericidal properties to aid in wound healing. Bandage wet necrotic wounds with a dressing premoistened with hypertonic saline (Curasalt [Kendall], 20% saline) to clean and debride the wounds. Hypertonic saline functions to desiccate necrotic tissue and bacteria to debride the infected wound. Remove and replace the hypertonic saline bandage every 24 to 48 hours. Next, place gauze impregnated with antibacterial agents (Kerlix AMD [Kendall]) over the wound in the bandage layer to act as a barrier to bacterial colonization.

If the wound is initially dry or has minimal exudate and is not obviously contaminated or infected, place amorphous gels of water, glycerin, and a polymer (Curafil [Kendall]) over the wound to promote moisture and proteolytic healing. Discontinue moisture gels such as Curafil once the dry wound has become moist.

Finally, the final stage of moist healing helps to promote the development of a healthy granulation tissue bed. Use calcium alginate dressings (Curasorb or Curasorb Zn with zinc [Kendall]) in noninfected wounds with a moderate amount of drainage. Alginate gels promote rapid development of a granulation tissue bed and epithelialization.

Foam dressings also can be applied to exudative wounds after a healthy granulation bed has formed. Change foam dressings at least once every 4 to 7 days.

CLOSED WOUNDS**Wounds with no drainage**

For closed wounds without any drainage, such as a laceration that has been repaired surgically, a simple bandage with a nonadherent contact layer (Telfa pad [Kendall], for example), intermediate layer of absorbent material, and an outer porous layer (Elastikon, Vetrap) can

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be placed to prevent wound contamination during healing. The nonadherent pad will not stick to the wound and cause patient discomfort. Because there usually is minimal drainage from the wound, the function of the intermediate layer is more protective than absorptive. Any small amount will be absorbed into the intermediate layer of the bandage. It is important in any bandage to place the tape strips or “stirrups” on the patient’s limb and then overlap in the bandage, to prevent the bandage from slipping. Place the intermediate and tertiary layers loosely around the limb, starting distally and working proximally, with some overlap with each consecutive layer. This method prevents excessive pressure and potential to impair venous drainage. Leave the toenails of the third and fourth digits exposed, whenever possible, to allow daily examination of the bandage to determine whether the bandage is impairing venous drainage. If the bandage is too tight and constricting or impeding vascular flow, the toes will become swollen and spread apart. When placed and maintained properly (e.g., the bandage does not get wet), there usually are relatively few complications observed with this type of bandage.

OPEN WOUNDS**Wounds with drainage**

In some cases, it is necessary to cover a wound in which a Penrose drain has been placed to allow drainage. In many cases, there is a considerable amount of drainage from the drain and underlying soft tissues. The function of the bandage is to help obliterate dead space created by the wound itself, absorb the fluid that drains from the wound and that will contaminate the environment, and prevent external wicking of material from the external environment into the wound. When the bandage is removed, the clinician can examine the amount and type of material that has drained from the wound in order to determine when the drain should be removed.

When placing a bandage over a draining wound, the contact layer should be a commercially available nonadherent dressing and several layers of absorbent wide-mesh gauze placed directly over the drain at the distal end of the incision. Overlay the layers of gauze with a thick layer of absorbent intermediate dressing to absorb fluid that drains from the wound. If the gauze and intermediate layers are not thick or absorbent enough, there is a potential for the drainage fluid to reach the outer layer of the bandage and provide a source of wicking of bacteria from the external environment into the wound, leading to infection.

WOUNDS IN NEED OF A PRESSURE BANDAGE**Minor hemorrhage**

Some wounds such as lacerations have minor bleeding or hemorrhage that require an immediate bandage until definitive care can be provided. To create a pressure bandage, place a nonadherent dressing immediately in contact with the wound, followed by a thick layer of absorbent material, topped by a layer of elastic bandage material such as Elastikon or Vetrup. Unlike the bandage for a closed wound, the top tertiary outer layer should be wrapped with some tension and even pressure around the limb, starting from the distal extremity (toes) and working proximally. The pressure bandage serves to control hemorrhage but should not be left on for long periods. Pressure bandages that have been left on for too long can impair nerve function and lead to tissue necrosis and slough. Therefore, pressure bandages should be used in the hospital only, so that the patient can be observed closely. If hemorrhage through the bandage occurs, place another bandage over the first until the wound can be repaired definitively. Removal of the first bandage will only disrupt any clot that has formed and cause additional hemorrhage to occur.

Initial fracture immobilization

Fractures require immediate immobilization to prevent additional patient discomfort and further trauma to the soft tissues of the affected limb. As with all bandages, a contact layer, intermediate layer, and outer layer should be used. Place the contact layer in accordance

with any type of wound present. The intermediate layer should be thick absorbent material, followed by a top layer of elastic bandage material. An example is to place a Telfa pad over a wound in an open distal radius-ulna fracture, followed by a thick layer of cotton gauze cast padding, followed by an elastic layer of Kling (Johnson & Johnson Medical, Arlington, Texas), pulling each layer tightly over the previous layer with some overlap until the resultant bandage can be “thumped” with the clinician’s thumb and forefinger and sound like a ripe watermelon. The bandage should be smooth with consecutive layers of even pressure on the limb, starting distally and working proximally. Leave the toenails of the third and fourth digits exposed to monitor for impaired venous drainage that would suggest that the bandage is too tight and needs to be replaced. Finally, place a top layer of Vetrap or Elastikon over the intermediary layer to protect it from becoming contaminated. If the bandage is used with a compound or open fracture, drainage may be impaired and actually lead to enhanced risk of wound infection. Bandages placed for initial fracture immobilization are temporary until definitive fracture repair can be performed once the patient’s cardiovascular and respiratory status are stable.

Exuberant granulation tissue

Wounds with exuberant granulation tissue must be handled carefully so as to not disrupt the healing process but to keep an overabundance of tissue from forming that will impair epithelialization. To bandage a wound with exuberant granulation tissue, place a corticosteroid-containing ointment on the wound, followed by a nonadherent contact layer. The corticosteroid will help control the exuberant growth of granulation tissue. Next, carefully wrap an absorbent material over the contact layer, followed by careful placement of and overlay of elastic bandage material to place some pressure on the wound. Leave the toenails of the third and fourth digits exposed so that circulation can be monitored several times daily. Bandages that are too tight must be removed immediately to prevent damage to neuronal tissue and impaired vascularization, tissue necrosis, and slough. Because wound drainage may be impaired, there is a risk of infection.

Obliteration of dead space

Gaping wounds or those that have undermined in between layers of subcutaneous tissue and fascia should be bandaged with a pressure bandage to help obliterate dead space and prevent seroma formation. An example of a wound that may require this type of bandage is removal of an infiltrative lipoma on the lateral or ventral thorax. Use caution when placing pressure bandages around the thorax or cervical region because bandages placed too tightly may impair adequate ventilation. To place a pressure bandage and obliterate dead space, place a nonadherent contact layer over the wound. Usually, a drain is placed in the wound, so place a large amount of wide-mesh gauze at the distal end of the drain to absorb any wound exudate or drainage. Place several layers of absorbent material over the site to further absorb any drainage. Place a layer of elastic cotton such as Kling carefully but firmly over the dead space to cause enough pressure to control drainage. Place at least two fingers in between the animal’s thorax and the bandage to ensure that the bandage is not too tight. In many cases, the bandage should be placed once the animal has recovered from surgery and is able to stand. If the bandage is placed while the animal is still anesthetized and recumbent, there is a tendency for the bandage to be too tight. Finally, the tertiary layer should be an elastic material such as Elastikon or Vetrap.

WOUNDS IN NEED OF PRESSURE RELIEF

Many wounds require a pressure relief bandage to prevent contact with the external environment. Wounds that may require pressure relief for healing include decubitus ulcers, pressure bandage or cast ulcers, impending ulcer areas (such as the ileum or ischium of recumbent or cachexic patients), and surgical repair sites of ulcerated areas. Pressure relief bandages can be of two basic varieties: modified doughnut bandage and doughnut-shaped bandage.

1

Modified doughnut bandage

A modified doughnut bandage should be placed over bony prominences on the limbs when there are early signs of pressure such as hyperemia, to prevent further injury. To place a modified doughnut bandage, cast padding material, thick wrapping material, and porous adhesive or loose elastic tape are required. Because this type of bandage becomes compressed after two to three bandage changes, it must be replaced frequently.

To place a modified doughnut bandage, follow this procedure (Figure 1-1):

1. Make several layers of cast padding, and fold them over together, making a 3 × 3-inch pad.
2. Fold the pad over on itself, and cut a slit in the center. Form this slit into a hole.
3. Place the hole in the cast padding over the bony prominence.
4. Wrap bandaging material over the pad.
5. Place tapes over the wrapping material, with overlap of the tape strips, to secure it in place.
6. Alternatively, place several loose stay sutures percutaneously in the skin surrounding the bony prominence, and secure the doughnut in place with umbilical tape woven through the stay sutures and over the doughnut.

Doughnut-shaped bandage

Like the modified doughnut-shaped bandage, a doughnut-shaped bandage is used over bony prominences to help prevent excessive pressure over the area. The bandage commonly is used over bony prominences on the distal limbs, such as the lateral malleolus, when more padding is indicated than is provided with a modified doughnut-shaped bandage. To make a doughnut-shaped bandage, use a hand towel or length of stockinet bandage material, tape, cotton gauze, elastic bandage material, or suture with umbilical tape. As the bandage becomes compressed or soiled, change it to prevent further damage to the underlying tissues.

To create a doughnut-shaped bandage, follow this procedure (Figures 1-2 and 1-3):

1. Roll a hand towel tightly and wrap tape around it to create a circle with a hole in the center. Alternatively, take a length of stockinet bandage material and roll it as you would a sock, creating a padded circle with a hole in the center. Make sure that the hole in the center is large enough to fit around the surgical repair site or ulcer.
2. Place the hole in the center over the ulcer or surgical repair site.
3. Secure the roll in place with strips of tape and cotton and then elastic bandage material. Alternatively, place loose loops of suture through the skin adjacent and around the wound. Secure the doughnut in place with umbilical tape secured through the suture loops and over the bandage. The wound in the center can be observed and treated through the hole in the center, if necessary.

WOUNDS IN NEED OF IMMOBILIZATION**External pin splints**

An external pin splint is required when fractures or luxations are associated with open wounds. In some cases, it may be difficult to bandage under the bars of the pin splint in such a way that the bandage is in contact with the wound. To create padding around the pins, fit foam rubber sponges to lie securely under and around the pins. Place bandage layers around the external fixator apparatus in layers to decrease contamination of the wound from the external environment and to absorb fluid that drains from the wound (Figures 1-4 to 1-6).

Cup or clamshell splints

A cup splint is indicated when bandaging pad wounds to decrease pressure on the footpad and prevent spreading of the footpads when the dog or cat places the paw down. If the toes spread, spreading of the footpad can delay or impair wound healing. The splint functions to place the paw in a more vertical direction so that the patient bears weight on the toe tips and not directly on the pads during the healing process.

Text continued on p. 18

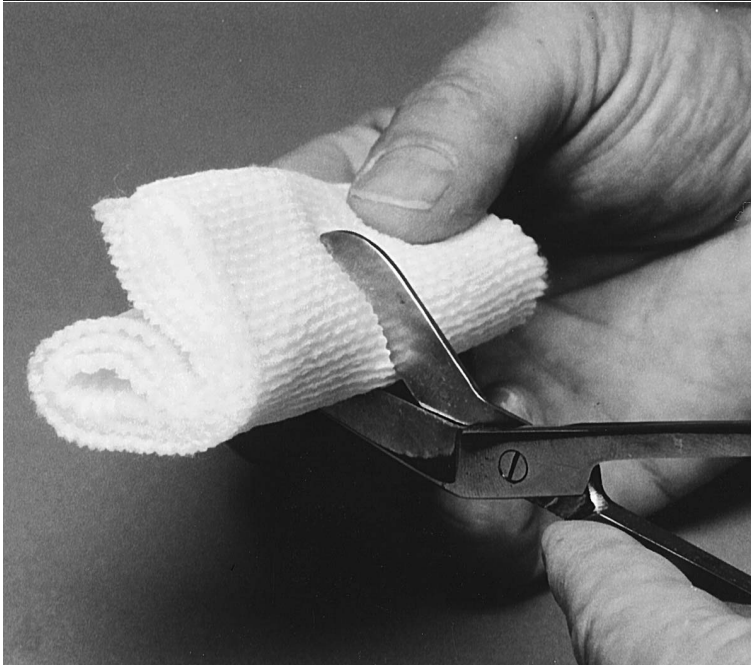
**A****B**

Figure 1-1: Modified doughnut bandage. **A,** Several layers of cast padding are folded together. **B,** The pad is folded on itself and a slit is cut in its center.

Continued

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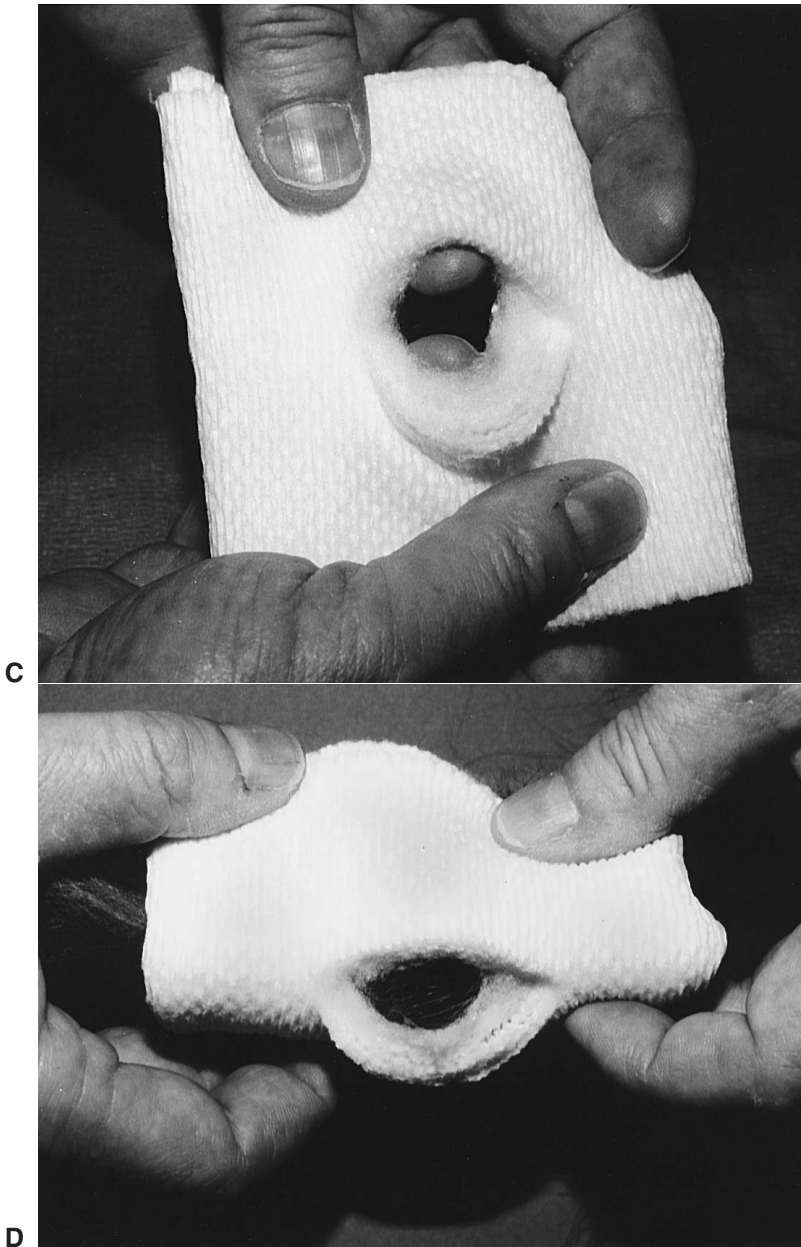


Figure 1-1, cont'd C, The slit is formed into a hole. D, The hole is placed over the bony prominence.

(From Swaim SE, Henderson RA: *Small animal wound management*. 2nd Edition. Williams & Wilkins, Media, Pa, 1997.)



Figure 1-2: Doughnut-shaped bandage created from stockinet bandage material over the olecranon.

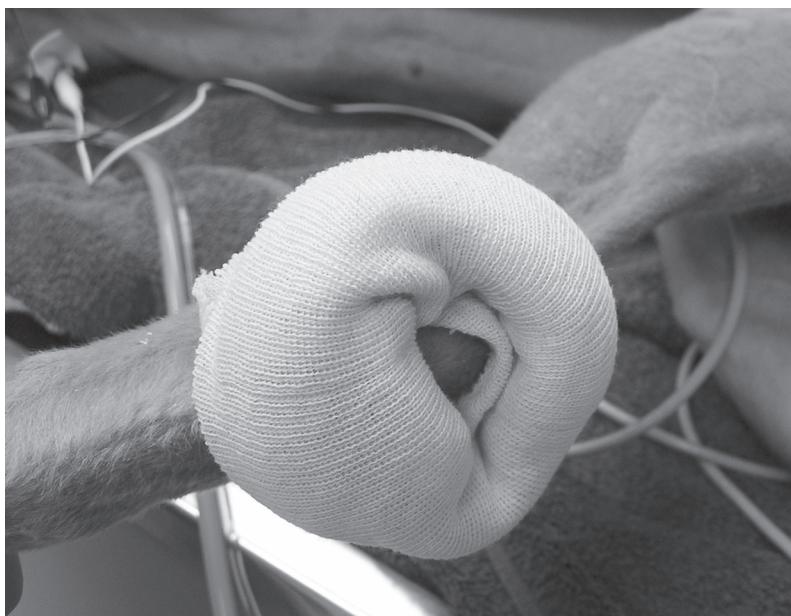


Figure 1-3: The tarsus.

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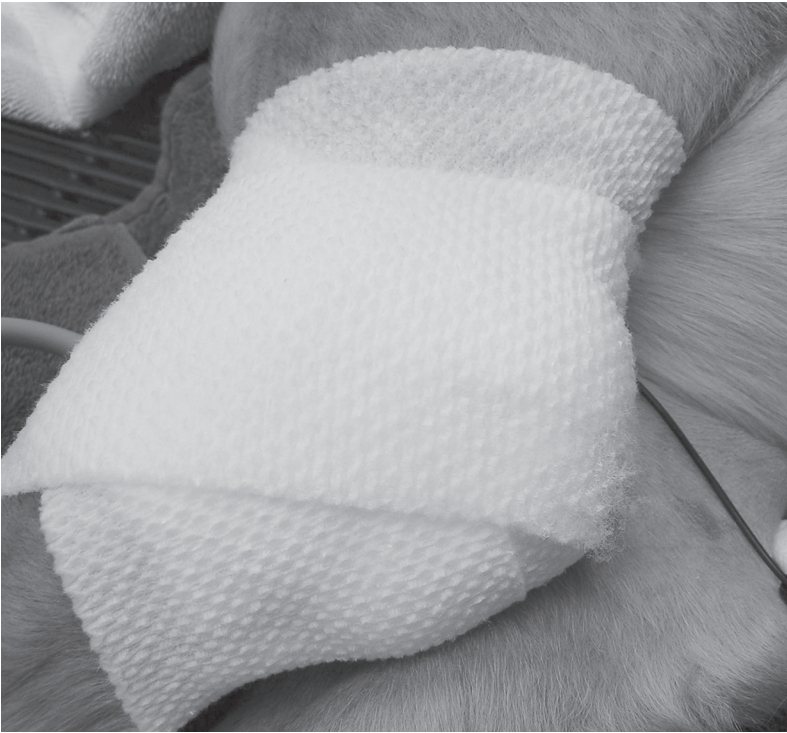


Figure 1-3, cont'd.

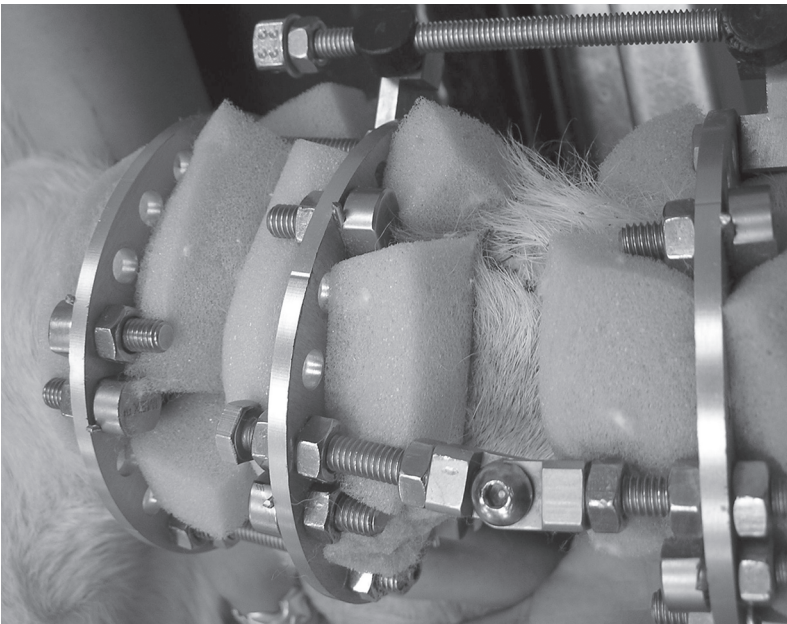


Figure 1-4: Foam rubber pads are placed under and around the pins of the external fixator, adjacent to the wound.



Figure 1-5: Cotton cast padding is placed around the external fixator to keep the foam rubber and contact layer securely in place.



Figure 1-6: Vetrap is placed over the intermediate layer to prevent contamination from the external environment.

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To create a cup or clamshell splint, follow this procedure (Figures 1-7 to 1-11):

1. Place a nonadherent contact layer directly over the wound.
2. Place stirrups of tape in contact with the skin of the dog, to be placed over the intermediate layer and prevent the bandage from slipping.
3. Place a fairly thick layer of absorbent intermediate bandage material over the contact layer such that the bandage is well-padded. Pull the tape stirrups and secure them to the intermediate layer.
4. Place a length of cast material that has been rolled to the appropriate length, such that the cast material is cupped around the patient's paw, and lies adjacent to the caudal aspect of the limb to the level of the carpus or tarsus. In the case of a clamshell splint, place a layer of cast material on the cranial and caudal aspect of the paw and conform it in place.



Figure 1-7: Tape stirrups in place.



Figure 1-8: Layer of absorbent roll cotton.



Figure 1-9: Secure tape stirrups to intermediate layer to prevent bandage from slipping.

5. Take the length of cast padding and soak it in warm water after it has been rolled to the appropriate length. Wring out the pad, and secure/conform it to the caudal (or cranial and caudal, in the case of a clamshell splint) aspect of the distal limb and paw.
6. Secure the cast material in place with a layer of elastic cotton gauze (Kling).
7. Secure the bandage in place with a snug layer of Elastikon or Vetrap.

Lateral or caudal splints

Short or long splints made of cast material can be incorporated into a soft padded bandage to provide extra support of a limb above and below a fracture site. For a caudal or lateral splint to be effective, it must be incorporated for at least one joint above any fracture site to prevent a fulcrum effect and further disruption or damage to underlying soft tissue structures. A short lateral or caudal splint is used for fractures and luxations of the distal metacarpus, metatarsus, carpus, and tarsus.

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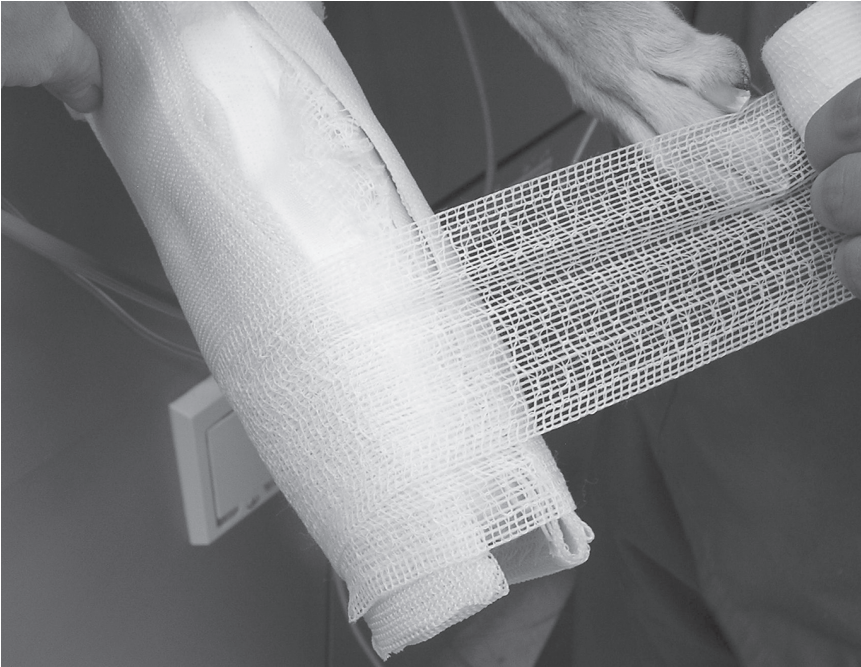


Figure 1-10: Place clamshell layer of cast material in place on the cranial and caudal aspect of the limb.

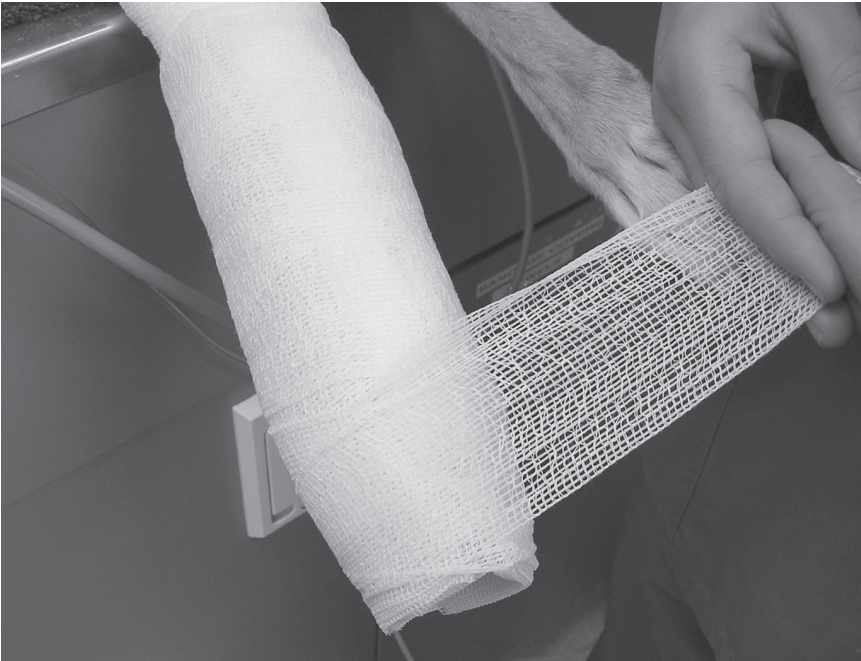


Figure 1-11: Secure the clamshell in place with Kling, then a layer of Vetrap.

To place a short lateral or caudal splint, follow this procedure:

1. Secure a contact layer as determined by the presence or absence of any wound in the area.
2. Place tape stirrups on the distal extremity to be secured later to the intermediate bandage layer and to prevent slipping of the bandage distally.
3. Place layers of roll cotton from the toes to the level of the mid tibia/fibula or mid radius/ulna. Place the layers with even tension, with some overlap of each consecutive layer, moving distally to proximally on the limb.
4. Secure the short caudal or lateral splint and conform it to the distal extremity to the level of the toes and proximally to the level of the mid tibia/fibula or mid radius/ulna.
5. Secure the lateral or caudal splint to the limb with another outer layer of elastic cotton (Kling).
6. Cover the entire bandage and splint with an outer tertiary layer of Vetrap or Elastikon. Make sure that the toenails of the third and fourth digits remain visible to allow daily evaluation of circulation.

Long lateral or caudal splints are used to immobilize fractures of the tibia/fibula and radius/ulna. The splints are fashioned as directed for short splints but extend proximally to the level of the axilla and inguinal regions to immobilize above the fracture site.

Spica splint

A spica splint is used to immobilize the humerus and elbow and shoulder joints in the case of luxation or fracture. To place a spica splint, follow this procedure:

1. Place a contact layer if any wounds are present.
2. Place tape stirrups to the distal limb to attach to the intermediate layer and prevent slipping of the bandage distally.
3. Place layers of conforming cotton gauze circumferentially and overlapping, moving up the limb from distal to proximal.
4. Incorporate the leg bandage into a layer of cotton bandage that is secured over the thorax.
5. Secure the cotton in place with a layer of snug elastic cotton material such as Kling. Make sure that the bandage is not so tight that breathing is impaired.
6. Place splint material on the lateral aspect of the limb, extending the material from the level of the toes proximally over the entire limb and extending proximally to over the scapula and dorsal midline.
7. After rolling the splint to an appropriate length and width, moisten the splint material in warm water to allow it to set and harden.
8. Replace the splint and conform it to the bandage over the patient's body.
9. Secure the splint in place with another layer of cotton Kling.
10. Wrap the entire bandage in place with a layer of tertiary bandage material such as Elastikon or Vetrap.

Additional Reading

Piermattei DL, Flo GL: *Brinker, Piermattei, and Flo's handbook of small animal orthopedics and fracture management*, ed 3, Philadelphia, 1997, WB Saunders.

BLOOD COMPONENT THERAPY

COLLECTION AND ADMINISTRATION

Blood component therapy involves the separation of blood into its cellular and fluid components and infusing the components specific for each patient's needs. Blood component therapy is the mainstay of initial and ongoing management of hematologic emergencies and can provide support of the critically ill patient until the underlying disease process is controlled. The separation of blood into red blood cells, plasma, cryoprecipitate, and platelet-rich products allows for more specific replacement of the animal's deficit(s),

BOX 1-4 APPROACH TO BLOOD COMPONENT THERAPY**RED BLOOD CELL SUPPORT**

- Packed cell volume drops rapidly to less than 20% in the dog and less than 12% to 15% in the cat
- Acute loss of more than 30% of blood volume (30 mL/kg in dog, 20 mL/kg in cat)
- Clinical signs of lethargy, collapse, hypotension, tachycardia, tachypnea (acute or chronic blood loss)
- Ongoing hemorrhage is present
- Poor response to crystalloid and colloid infusion

PLATELET SUPPORT

- Life-threatening hemorrhage caused by thrombocytopenia or thrombocytopathia
- Surgical intervention is necessary in a patient with severe thrombocytopenia or thrombocytopathia

PLASMA SUPPORT

- Life-threatening hemorrhage with decreased coagulation factor activity
- Severe inflammation (pancreatitis, systemic inflammatory response syndrome)
- Replenish antithrombin (disseminated intravascular coagulation, protein-losing enteropathy or nephropathy)
- Surgery is necessary in a patient with decreased coagulation factor activity
- Severe hypoproteinemia is present; to partially replenish albumin, globulin, and clotting factors

decreases the risks of transfusion reactions, and allows for more efficient use of donor blood. Box 1-4 lists indications for transfusion of red blood cells, platelet-rich plasma, fresh frozen or fresh plasma, and cryoprecipitate.

BLOOD TYPES AND ANTIGENICITY

Cell membrane receptors on the surface of red blood cells (RBCs) serve the purpose of self-recognition versus non-self-recognition during states of health. The presence or absence of various glycoprotein and glycolipid moieties on the RBC surface helps to define blood groups or “types” within a species. In dogs, six cell surface dog erythrocyte antigens (DEAs, 1.1, 1.2, 3, 4, 5, and 7) have been identified. Dogs that are negative for DEA 1.1, 1.2, and 7 but positive for DEA 1.4 are known as universal donors and have type A-negative blood. Dog erythrocyte antigens 1.1 and 1.2 are the most immunogenic RBC antigens known in canine transfusion medicine. Transfusion of DEA 1.1- or 1.2-positive blood to a DEA 1.1- and 1.2-negative dog can result in immediate hemolysis or a delayed-type hypersensitivity reaction. Additionally, viability of DEA 1.1- and 1.2-positive cells in a DEA 1.1- and 1.2-negative recipient is short-lived, ultimately defeating the long-term goal of increasing oxygen delivery in the recipient.

Like dogs, feline blood groups are defined by specific carbohydrate moieties attached to lipids (glycolipids) and proteins (glycoproteins) on the RBC surface. Three blood types (A, B, and AB) have been identified in cats. Type A is the most common blood type in cats. Type B is relatively uncommon and occurs in Abyssinian, Persian, Devon Rex, and British Shorthair cats but can be found in domestic shorthair and longhair cats as well. Type A is completely dominant over type B by simple mendelian genetics. Type AB is a rare blood type that has been identified infrequently in domestic short-haired cats, Birman, Abyssinian, Somali, British Shorthair, Scottish Fold, and Norwegian Forest Cats. Unlike dogs, cats possess naturally occurring antibodies against other feline blood types. The presence of naturally occurring autoantibodies is of paramount importance, necessitating blood typing with or without crossmatch before any feline transfusion, because hemolytic transfusion reactions potentially can be fatal, even with no prior sensitization or blood transfusion. Type B cats possess large quantities of anti-A antibodies, primarily of the immunoglobulin M (IgM) subclass. Type A blood infused into a type B cat will be destroyed within minutes to hours, and as little as 1 mL of incompatible blood can cause a life-threatening reaction.

Type A cats typically possess weak anti-B antibodies of IgG and IgM subtypes. Transfusion of type B blood into a type A cat will result in milder clinical signs of reaction and a markedly decreased survival half-life of the infused RBCs to just 2 days. Because type AB cats possess both moieties on their cell surface, they lack naturally occurring alloantibodies; transfusion of type A blood into a type AB cat can be performed safely if a type AB donor is not available. The life span of an RBC from a type-specific transfusion into a cat is approximately 33 days.

BLOOD DONORS PROGRAMS

Each clinic must weigh the cost-benefit ratio, the need for blood products, and the overall quantity of blood products in the practice when deciding which option works best for the staff, clientele, and patient needs. Busy hospitals requiring large quantities of blood products at regular intervals may elect to keep an in-house colony of donor dogs and cats. Maintenance of a closed donor colony may be impractical because of the economics of feeding and housing the animals and using cage space that can be used for other patients. Additionally, care of the animals—including frequent health examinations, blood testing (complete blood count, biochemistry panels, heartworm tests), and daily care—are labor intensive for veterinarian and support staff alike. Other options include using staff- or client-owned animals as donors. This practice eliminates the expense of housing donors within the clinic and the labor required for daily care. Donor animals can be used as needed or can have scheduled collections to replenish the stock of blood products. The final option, which may be more practical for clinics with an infrequent need for blood products, is to purchase blood components from a commercial blood bank (Table 1-1).

Blood donors should receive annual physical examinations and general health screens, including a complete blood count, serum biochemistry panel, and occult heartworm antigen test. Canine donors also should be screened initially for Lyme disease, *Babesia*, Rocky Mountain spotted fever (*Rickettsia rickettsii*), *Ehrlichia*, and *Brucella*. The prevalence of *Babesia* spp. in the racing Greyhound industry in Florida, Arizona, and Colorado is high (estimated to be 30% to 50%). Dogs ideally should weigh greater than 50 lb (27 kg), be between 1 and 8 years of age, have a packed cell volume (PCV) of at least 40%, be spayed

TABLE 1-1 List of Veterinary Blood Banks

Name/Address	Telephone number	Web site
Animal Blood Bank P.O. Box 1118 Dixon, CA 95620	800-243-5759 (800-2HELPK9)	www.animalbloodbank.com/
Eastern Veterinary Blood Bank 844 Ritchie Highway Suite 204 Severna Park, MD 21146	800-949-EVBB (800-949-3822)	www.evbb.com
Hemopet Blood Bank Office 11330 Markon Drive Garden Grove, CA 92841	714-891-2022	www.itsfortheanimals.com/HEMOPET.HTM
Midwest Animal Blood Services 4983 Bird Drive Stockbridge, MI 49285	877-517-MABS (877-517-6227)	www.midwestabs.com

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and/or nulliparous, and have never received a transfusion. A healthy donor safely can donate 10 to 20 mL/kg of whole blood every 3 to 4 weeks if necessary.

Feline blood donors ideally should weigh greater than 8 lb, be between 1 and 8 years of age, be nulliparous and/or spayed, and have never received a transfusion. Additionally, donor cats should be screened for feline leukemia virus, feline immunodeficiency virus, *Hemobartonella*, *Mycoplasma haemofelis* and feline infectious peritonitis before donations and should have a minimally acceptable PCV of 30%, although between 35% and 40% is preferred. Whole blood and plasma from donors that previously have been pregnant or received a transfusion should not be used because of the risk of previous exposure to foreign RBC antigens and the development of antibodies.

BLOOD COLLECTION AND HANDLING

Any blood collection should be performed in a manner that is the least stressful for the donor animal. Physical examination and determination of PCV and total solids should be performed before any donation. Blood can be obtained from a jugular vein or femoral artery. However, because of the risk of lacerating the femoral artery, with subsequent hemorrhage or development of compartmental syndrome, I strongly advocate using the jugular vein as the primary site of blood collection in dogs and cats. Carefully clip the fur over the jugular vein, avoiding skin abrasions. Place dogs in lateral recumbancy; however, sternal recumbancy or sitting on the floor are also acceptable methods. Aseptically scrub the area over the clipped jugular vein with iodine or chlorhexidine wipes, alternating with alcohol or sterile saline. Blood can be collected from an open or closed system. Closed-system collection is preferred because it decreases the potential for contamination of the blood product and facilitates processing of blood components. Alternatively, an open system can be used if the blood is going to be transfused within 24 hours. Gently insert a 16-gauge needle into the jugular vein. Place the collection system on a scale on the floor and zero the scale. Then remove the hemostat placed on the collection tubing and allow the blood to flow by gravity. Canine units should be approximately 450 mL, which translates to 450 g on the tared scale, because 1 mL weighs approximately 1 g. Although a volume of 450 mL can be obtained every 21 days, if necessary, from a healthy canine donor, less frequent donation of every 3 months is preferred.

Alternatively, feline blood collection often requires the use of sedation, unless a multi-access port has been implanted surgically. All donor cats should have a physical examination and determination of PCV and total solids performed before sedation and subsequent blood donation. Clip the fur over the jugular vein and aseptically prepare the site as described previously. Insert a 19-gauge butterfly catheter into the jugular vein and aspirate blood with gentle pressure to avoid venous collapse. The butterfly catheter is attached to a three-way stopcock and 60-mL syringe into which 7 mL of citrate phosphate dextrose adenine anticoagulant has been placed. In most cases, a total volume of 53 mL of blood is obtained. The blood can be transfused immediately or placed into a small sterile collection bag that contains 0.14 mL of citrate phosphate dextrose adenine anticoagulant per milliliter of whole blood. No more than 11 to 15 mL/kg should be obtained at any given time from a feline donor (Box 1-5).

BOX 1-5 SUPPLIES NEEDED FOR BLOOD COLLECTION AND PROCESSING

CANINE	FELINE
Blood donor collection bag	60-mL syringe
Sealing clips	Three-way stopcock
Pliers or tube stripper (optional)	7 mL citrate phosphate dextrose adenine anticoagulant solution
Guarded hemostat	Ketamine
Plasma press	Diazepam

BLOOD COMPONENT PROCESSING AND STORAGE

Production of blood component therapy has become more commonplace in human and veterinary medicine. Component therapy involves the separation of whole blood into its cellular and plasma components and then administration of specific components to a recipient based on each patient's individual needs. Preparation of fresh frozen plasma, frozen plasma, cryoprecipitate, and cryopoor plasma requires the use of a refrigerated centrifuge. Floor and tabletop models are currently available for purchase. In many cases, purchase of a refrigerated centrifuge is impractical because of the expense and the space required for its storage. A veterinary community potentially can pool resources for the cost of the equipment and house the unit at a centrally located facility, such as a local emergency hospital. Alternatively, human hospitals or blood banks may provide separation services for a nominal fee. Investigation of guidelines in your area may provide a means of creating blood components from your donors for use in your practice.

Once obtained, blood should be stripped from the collection tubing, and the line sealed using a thermal seal or aluminum clips. The bag should be labeled clearly with donor name, donor blood type, date of collection, PCV or donor at time of collection, and date of expiration. If the blood is not going to be used immediately or prepared for platelet-rich plasma, the unit should be refrigerated. The unit then can be spun at 4000 to 5000 times gravity for 5 minutes to separate the RBCs from plasma components. Use of a plasma extractor will facilitate flow of plasma into designated satellite bags for further storage.

Fresh frozen plasma, cryoprecipitate, and cryopoor plasma should be frozen within 8 hours of collection to ensure preservation of labile clotting factors, including factors V and VIII and von Willebrand's factor (vWF). Fresh frozen plasma has a shelf life of 1 year past the date of collection. Before freezing, place an elastic band around the bag to crimp the bag during the freezing process. The elastic band is removed once the unit is frozen. In case of an inadvertent or unobserved power failure, the crimp in the unit provides a quality control measure that inadvertent thawing has not occurred. Partial thawing and differential centrifugation of fresh frozen plasma allows preparation of cryoprecipitate and cryopoor plasma. Following 1 year, or if a unit of plasma has been prepared after 8 hours of collection, frozen plasma results. Frozen plasma contains all of the vitamin K–dependent coagulation factors (II, VII, IX, X), immunoglobulins, and albumin but is relatively devoid of the labile clotting factors. Frozen plasma has a shelf life of 5 years after the original date of collection or 4 years after expiration of a unit of fresh frozen plasma. Packed RBCs should be stored at 1 to 6° C immediately after collection and processing. Packed RBCs and frozen plasma also can be prepared in the absence of a refrigerated centrifuge by storing the unit of whole blood upright in a refrigerator at 1 to 6° C for 12 to 24 hours until the RBCs have separated out. The plasma can be drawn off into a second storage bag and frozen as frozen plasma. Because of the delay in processing, the resultant plasma does not contain the labile clotting factors. Fresh frozen plasma, frozen plasma, cryoprecipitate, and cryopoor plasma should be stored at –20° C until use (Table 1-2). The products should be thawed in tepid water until no crystals are observed. No plasma product should be heated to greater than 37° C because protein denaturation can occur.

Crossmatch procedure

Before administering blood products, find out the donor and recipient's blood types and perform a crossmatch procedure as time allows. At minimum, a blood type should be performed before administration. Rapid blood typing cards are available for use in dogs and cats (Rapid Vet-H; DMS Laboratories, Flemington, New Jersey). A crossmatch procedure simulates in vitro the response of a recipient to donor plasma and RBC antigens. The crossmatch procedure is performed to decrease the risk of transfusion reactions in patients that have been sensitized previously, that have naturally occurring alloantibodies, or in situations of neonatal isoerythrolysis. Other indications for crossmatching include decreasing the risk of sensitizing a patient if more than one transfusion is anticipated. Crossmatching can be divided into major and minor categories. The major crossmatch mixes the donor's

TABLE 1-2 Storage of Blood Components

Component	Anticoagulant	Shelf life	Comments
Whole blood (WB)	Heparin 625 IU/ 250 mL WB	37 days, 4° C	No preservation, coagulation factor inhibition
	ACD, 10 mL/ 60 mL WB	24 hours, 4° C	ACD rarely used
	CPDA-1, 1 mL/ 7 mL WB	21 days, 4° C	Will maintain 75% PTV
	AS-1	35 days, 4° C	
Packed RBCs	CPDA-1	20 days, 4° C	Will maintain 75% PTV
	AS-1	37 days, 4° C	
Platelet-rich plasma	CPDA-1	3-5 days, 23° C	Needs constant agitation
		2 hours, 4° C	
Fresh frozen plasma	CPDA-1	1 year, -30° C	Frozen <6 hours after collection of blood; all coagulation factors present
		3 months, -18° C	
Plasma/cryopoor plasma	CPDA-1	5 years, -30° C	Does not contain factors V and VIII
Cryoprecipitate	CPDA-1	1 year, -30° C	High concentration of vWF, factor VIII, and fibrinogen

ACD, Acid citrate dextrose; AS, additive solution; CPDA-1, citrate phosphate dextrose adenine; PTV, posttransfusion viability; RBCs, red blood cells; vWF, von Willebrand's factor; WB, whole blood.

RBCs with recipient's plasma, thus testing whether the recipient contains antibodies against donor RBCs. A minor crossmatch mixes donor plasma with recipient RBCs, testing for the unlikely occurrence that the donor serum contains antibodies directed against recipient RBCs. Box 1-6 gives a complete step-by-step description of major and minor crossmatch procedures. The crossmatch procedures do not check for other sources of immediate hypersensitivity transfusion reactions, including white blood cell and platelets.

INDICATIONS FOR TRANSFUSION THERAPY

There are many indications for administering transfusions of whole blood and component blood products. Take a stepwise approach for every patient that may require a transfusion. If a patient is at risk for blood loss or is anemic, consider a transfusion. Make a decision on the type of transfusion therapy appropriate for each particular patient. Once a decision is made about which components need to be administered, calculate a volume to be delivered. Exercise caution when administering larger volumes to small patients or those with cardiac insufficiency, because volume overload potentially can occur. If RBC products are to be administered, a minimum of a blood type should be performed before giving type-specific blood. The gold standard is to perform a crossmatch for each unit administered to decrease the risk of a transfusion reaction or sensitizing the patient to foreign RBC antigens. In patients with severe hemorrhage when there is not enough time even for performing a blood type, universal blood (DEA 1.1-, 1.2-, and 1.7-negative) or Oxyglobin (Biopure, Cambridge, Massachusetts) can be administered.

A common misconception is that administration of whole blood or packed RBCs should occur when patient PCV decreases to a certain number. In fact, no absolute "transfusion trigger" number actually exists. Administer a transfusion whenever a patient demonstrates clinical signs of anemia, including lethargy, anorexia, weakness, tachycardia, and/or tachypnea

BOX 1-6 PROTOCOL FOR PERFORMING MAJOR AND MINOR CROSSMATCH

Supplies needed: 0.9% physiologic saline in wash bottle

3-mL test tubes

Pasteur pipettes

Centrifuge

Agglutination viewer lamp

1. Label test tubes as follows:

RC Recipient control

RR Recipient RBCs

RP Recipient plasma

DB Donor whole blood*

DC Donor control*

DR Donor whole blood*

DP Donor plasma*

Ma Major crossmatch*

Mi Minor crossmatch*

2. Obtain a crossmatch segment from blood bank refrigerator for each donor to be cross-matched, or use an EDTA tube of donor's blood. **MAKE SURE TUBES ARE LABELED PROPERLY.**

3. Collect 2 mL of blood from recipient and place in an EDTA tube. Centrifuge blood for 5 minutes.

4. Extract blood from donor tubing. Centrifuge blood for 5 minutes. Use a separate pipette for each transfer because cross-contamination can occur.

5. Pipette plasma off of donor and recipient cells and place in tubes labeled DP and RP, respectively.

6. Place 125 μ L of donor and recipient cells in tubes labeled DR and RR, respectively.

7. Add 2.5 mL 0.9% sodium chloride solution from wash bottle to each red blood cell (RBC) tube, using some force to cause cells to mix.

8. Centrifuge RBC suspension for 2 minutes.

9. Discard supernatant and resuspend RBCs with 0.9% sodium chloride from wash bottle.

10. Repeat steps 8 and 9 for a total of three washes.

11. Place 2 drops of donor RBC suspension and 2 drops of recipient plasma in tube labeled Ma (this is the major crossmatch).

12. Place 2 drops of donor plasma and 2 drops recipient RBC suspension in tube labeled Mi (this is the minor crossmatch).

13. Prepare control tubes by placing 2 drops donor plasma with 2 drops donor RBC suspension (this is the donor control); and place 2 drops recipient plasma with 2 drops recipient RBC suspension (this is the recipient control).

14. Incubate major and minor crossmatches and control tubes at room temperature for 15 minutes.

15. Centrifuge all tubes for 1 minute.

16. Read tubes using an agglutination viewer.

17. Check for agglutination and/or hemolysis.

18. Score agglutination with the following scoring scale:

4+ One solid clump of cells

3+ Several large clumps of cells

2+ Medium-sized clumps of cells with a clear background

1+ Hemolysis, no clumping of cells

NEG = Negative for hemolysis; negative for clumping of red blood cells

*Indicates that this must be done for each donor being tested.

(Table 1-3). Indications for fresh whole blood transfusion include disorders of hemostasis and coagulopathies including disseminated intravascular coagulation, von Willebrand's disease, and hemophilia. Fresh whole blood and platelet-rich plasma also can be administered in cases of severe thrombocytopenia and thrombocytopathia. Stored whole blood and packed RBCs can be administered in patients with anemia. If PCV drops to below 10% or if rapid hemorrhage causes the PCV to drop below 20% in the dog or less than 12% to

TABLE 1 - 3 Indications for Administration of Blood Products

Blood products	Indications
Fresh whole blood	Coagulopathy with active hemorrhage (disseminated intravascular coagulation, thrombocytopenia; massive acute hemorrhage; no stored blood available)
Stored whole blood	Massive acute or ongoing hemorrhage; hypovolemic shock caused by hemorrhage that is unresponsive to conventional crystalloid and colloid fluid therapy; unavailability of equipment required to prepare blood components
Packed red blood cells	Nonregenerative anemia, immune-mediated hemolytic anemia, correction of anemia before surgery, acute or chronic blood loss
Fresh frozen plasma	Factor depletion associated with active hemorrhage (congenital: von Willebrand's factor, hemophilia A, hemophilia B; acquired: vitamin K antagonist, rodenticide intoxication, DIC); acute or chronic hypoproteinemia (burns, wound exudates, body cavity effusion; hepatic, renal, or gastrointestinal loss); colostrum replacement in neonates
Frozen plasma (contains stable clotting factors)	Acute plasma or protein loss; chronic hypoproteinemia; colostrum replacement in neonates; hemophilia B and selected clotting factor deficiencies
Platelet-rich plasma*	Thrombocytopenia with active hemorrhage (immune-mediated thrombocytopenia, DIC); platelet function abnormality (congenital: thrombasthenia in Bassett hounds; acquired: NSAIDs, other drugs)
Cryoprecipitate (concentration of factor VIII, von Willebrand's factor, and fibrinogen)	Congenital factor deficiencies (routine or before surgery): hemophilia A, hemophilia B, von Willebrand's disease, hypofibrinogenemia; acquired factor deficiencies

*Must be purchased because logistically one cannot obtain enough blood simultaneously to provide a significant amount of platelets; platelets infused have a very short (<2 hours) half-life.
DIC, Disseminated intravascular coagulation; NSAIDs, nonsteroidal antiinflammatory drugs.

15% in the cat, a transfusion is advocated. Consider fresh frozen plasma or cryoprecipitate administration in cases of coagulopathy, including von Willebrand's disease, rodenticide intoxication with depletion of activated vitamin K–dependent coagulation factors, and hemophilia or in cases of severe hypoproteinemia with albumin concentrations less than 2.0 g/dL. Frozen plasma also will suffice in cases of severe hypoproteinemia, warfarin-like compound intoxication, and factor IX deficiency (hemophilia B).

CONSIDERATIONS FOR ADMINISTRATION OF BLOOD COMPONENT THERAPY

When considering the type of blood component product required for transfusion, one should answer a number of questions to decrease the risk of a transfusion reaction and to decrease the risk for rejection or destruction of the component that has been infused. First, knowledge of a patient's blood type is essential. Whenever possible, type-specific RBCs should be administered. If an animal has received prior transfusion(s), the risk of a transfusion reaction or rejection is increased because of the development of antibodies directed against glycoprotein moieties on the surface of RBCs. If a prior transfusion has taken place, the patient's blood (RBCs and plasma) must be crossmatched with the donor blood (RBCs and plasma) to make sure that no incompatibility exists. In dogs, if neither blood typing nor crossmatch procedure is available, or if the emergent situation requires that a transfusion be administered before a blood type or crossmatch can be performed, blood from a

TABLE 1-4 Blood Component Dose and Administration Rates

Component	Dose	Administration rate	
		Normovolemia	Hypovolemia
Whole blood	20 mL/kg will increase volume by 10%	Max rate: 22 mL/kg/24 hours	Max: 22 mL/kg/hour
Packed red blood cells	10 mL/kg will increase volume by 10%		Critically ill patients (e.g., cardiac failure or renal failure): 3-4 mL/kg/hour
Fresh frozen plasma	10 mL/kg body mass (repeat in 2-3 days or in 3-5 days or until bleeding stops); monitor ACT, APTT, and PT before and 1 hour after transfusion	4-10 mL/minute or use whole blood (infuse within 4-6 hours)	rates as for whole blood
Cryoprecipitate	General: 1 unit/10 kg/12 hours or until bleeding stops Hemophilia A: 12-20 units factor VIII/kg; 1 unit of cryoprecipitate contains approximately 125 units of factor VIII	4-10 mL/minute or use whole blood (infuse within 4-6 hours)	
Platelet-rich plasma	1 unit/10 kg (1 unit of platelet-rich plasma will increase platelet count 1 hour after transfusion by 10,000/ μ L)	2 mL/minute Check platelet count before and 1 hour after transfusion	

ACT, Activated clotting time; APTT, activated partial thromboplastin time; PT, prothrombin time.

universal donor (e.g., DEA 1.1-, 1.2-, and 1.7-negative) should be administered whenever possible. Because there is no universal donor in the cat and because cats possess naturally occurring alloantibodies, all cat blood should be typed and crossmatched before any transfusion. If fresh whole blood is not available, a hemoglobin-based oxygen carrier (Oxyglobin, 2 to 7 mL/kg IV) can be administered until blood products become available.

TRANSFUSION OF BLOOD COMPONENT PRODUCTS

Table 1-4 indicates blood component dose and administration rates.

Red blood cell component therapy

Blood products should be warmed slowly to 37° C before administering them to the patient. Blood warmer units are available for use in veterinary medicine to facilitate rapid transfusion without decreasing patient body temperature (Thermal Angel; Enstill Medical Technologies, Inc., Dallas, Texas). Red blood cell and plasma products should be administered in a blood administration set containing a 170- μ m in-line filter. Smaller in-line filters (20 μ m) also can be used in cases in which extremely small volumes are to be administered. Blood products should be administered over a period of 4 hours, whenever possible, according to guidelines set by the American Association of Blood Banks.

The volume of blood components required to achieve a specific increment in the patient's PCV depends largely on whether whole blood or packed RBCs are transfused and

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whether there is ongoing hemorrhage or RBC destruction. Because the PCV of packed RBCs is unusually high (80% for Greyhound blood), a smaller total volume is required than whole blood to achieve a comparable increase in the patient's PCV. In general, 10 mL/kg of packed RBCs or 20 mL/kg whole blood will raise the recipient's PCV by 10%. The "Rule of Ones" states that 1 mL per 1 lb of whole blood will raise the PCV by 1%. If the patient's PCV does not raise by the amount anticipated by the foregoing calculation(s), causes of ongoing hemorrhage or destruction should be considered. The goal of red blood component therapy is to raise the PCV to 25% to 30% in dogs and 15% to 20% in cats.

If an animal is hypovolemic and whole blood is administered, the fluid is redistributed into the extravascular compartment within 24 hours of transfusion. This will result in a secondary rise in the PCV 24 hours after the transfusion in addition to the initial rise 1 to 2 hours after the RBC transfusion is complete.

Use of fresh frozen plasma

The volume of plasma transfused depends largely on the patient's need. In general, plasma transfusion should not exceed more than 22 mL/kg during a 24-hour period for normovolemic animals. Thaw plasma at room temperature, or place it in a ziplock freezer bag and run under cool (not warm) water until thawed. Then administer the plasma through a blood administration set that contains an in-line blood filter or through a standard drip-type administration set with a detachable in-line blood administration filter. The average rate of plasma infusion in a normovolemic patient should not exceed 22 mL/kg/hour. In acute need situations, plasma can be delivered at rates up to 5 to 6 mL/kg/minute. For patients with cardiac insufficiency or other circulatory problems, plasma infusion rates should not exceed 5 mL/kg/hour. Plasma or other blood products should not be mixed with or used in the same infusion line as calcium-containing fluids, including lactated Ringer's solution, calcium chloride, or calcium gluconate. The safest fluid to mix with any blood product is 0.9% sodium chloride.

Administer fresh frozen plasma, frozen plasma, and cryoprecipitate at a volume of 10 mL/kg until bleeding is controlled or source of ongoing albumin loss ceases. The goal of plasma transfusion therapy is to raise the albumin to a minimum of 2.0 g/dL or until bleeding stops as in the case of coagulopathies. Monitor the patient to ensure that bleeding has stopped, coagulation profiles (ACT, APTT, and PT) have normalized, hypovolemia has stabilized, and/or total protein is normalizing, which are indications for discontinuing ongoing transfusion therapy.

Use of plasma cryoprecipitate

Plasma cryoprecipitate can be purchased or manufactured through the partial thawing and then centrifugation of fresh frozen plasma. Cryoprecipitate contains concentrated quantities of vWF, factor VIII, and fibrinogen and is indicated in severe forms of von Willebrand's disease and hemophilia A (factor VIII deficiency).

Platelet-rich plasma

Platelet-rich plasma must be purchased from a commercial source. One unit of fresh whole blood contains 2000 to 5000 platelets. The viability of the platelets contained in the fresh whole blood is short-lived, just 1 to 2 hours after transfusion into the recipient. Because platelet-rich plasma is difficult to obtain, animals with severe thrombocytopenia or thrombocytopathia should be treated with immunomodulating therapies and the administration of fresh frozen plasma.

TRANSFUSION IN DOGS

In dogs, blood and plasma transfusions can be administered intravenously or intraosseously. The cephalic, lateral saphenous, medial saphenous, and jugular veins are used most commonly. Fill the recipient set so that the blood in the drip chamber covers the filter (normal 170- μ m filter). With small amounts of blood (50 mL) or critically ill patients, use a 40- μ m filter. Avoid latex filters for plasma and cryoprecipitate administration. Blood can

be administered at variable rates, but the routine figure of 4 to 5 mL/minute often is used. Normovolemic animals can receive blood at 22 mL/kg/day. Dogs in heart failure should receive infusions at no more than 4 mL/kg/hour. Volume is given as needed. To calculate the approximate volume of blood needed to raise hematocrit levels, use the following formula for the dog:

$$\begin{aligned} &\text{Anticoagulated blood volume (mL)} \\ &= \text{Body mass (kg)} \times 90 \times \frac{\text{PCV desired} - \text{PCV of recipient}}{\text{PCV of donor in anticoagulant}} \end{aligned}$$

An alternative formula is the following:

$$2.2 \times \text{Recipient body mass (kg)} \times 30 \text{ (dog)} \times \frac{\text{PCV desired} - \text{PCV of recipient}}{\text{PCV of donor in anticoagulant}}$$

Surgical emergencies and shock may require several times this volume within a short period. If greater than 25% of the patient's blood volume is lost, supplementation with colloids, crystalloids, and blood products is indicated for fluid replacement. One volume of whole blood achieves the same increase in plasma as two to three volumes of plasma. If the patient's blood type is unknown and type A-negative whole blood is not available, any dog blood can be administered to a dog in acute need if the dog has never had a transfusion before. If mismatched blood is given, the patient will become sensitized, and after 5 days, destruction of the donor RBCs will begin. In addition, any subsequent mismatched transfusions may cause an immediate reaction (usually mild) and rapid destruction of the transfused RBCs.

The clinical signs of a transfusion reaction typically only are seen when type A blood is administered to a type A-negative recipient that has been sensitized previously. Incompatible blood transfusions to breeding females can result in isoimmunization and in hemolytic disease in the puppies. The A-negative bitch that receives a transfusion with A-positive and that produces a litter from an A-positive stud can have puppies with neonatal isoerythrolysis.

TRANSFUSION IN CATS

Cats with severe anemia in need of a blood transfusion are typically extremely depressed, lethargic, and anorexic. The stress of restraint and handling can push these critically ill patients over the edge and cause them to die. Extreme gentleness and care are mandatory in restraint and handling. The critically ill cat should be cradled in a towel or blanket. Supplemental flow-by or mask oxygen should be administered, whenever possible, although it may not be clinically helpful until oxygen-carrying capacity is replenished with infusion of RBCs or hemoglobin.

Blood can be administered by way of cephalic, medial saphenous, or the jugular vein. Intramedullary infusion is also possible, if vascular access cannot be accomplished. The average 2- to 4-kg cat can accept 40 to 60 mL of whole blood injected intravenously over a period of 30 to 60 minutes. Administer filtered blood at a rate of 5 to 10 mL/kg/hour. The following formula can be used to estimate the volume of blood required for transfusion in a cat:

$$\begin{aligned} &\text{Anticoagulated blood volume (mL)} \\ &= \text{Body mass (kg)} \times 70 \times \frac{\text{PCV desired} - \text{PCV of recipient}}{\text{PCV of donor in anticoagulant}} \end{aligned}$$

TRANSFUSION REACTIONS

The exact overall incidence and clinical significance of transfusion reactions in veterinary medicine are unknown. Several studies have been performed that document the incidence of transfusion reactions in dogs and cats. Overall, the incidence of transfusion reactions in

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dogs and cats is 2.5% and 2%, respectively. Transfusion reactions can be immune-mediated and non-immune-mediated and can happen immediately or can be delayed until after a transfusion. Acute reactions usually occur within minutes to hours of the onset of transfusion but may occur up to 48 hours after the transfusion has been stopped. Acute immunologic reactions include hemolysis and acute hypersensitivity including RBCs, platelets, and leukocytes. Signs of a delayed immunologic reaction include hemolysis, purpura, immunosuppression, and neonatal isoerythrolysis. Acute nonimmunologic reactions include donor cell hemolysis before onset of transfusion, circulatory volume overload, bacterial contamination, citrate toxicity with clinical signs of hypocalcemia, coagulopathies, hyperammone-mia, hypothermia, air embolism, acidosis, and pulmonary microembolism. Delayed nonimmunologic reactions include the transmission and development of infectious diseases and hemosiderosis. Clinical signs of a transfusion reaction typically depend on the amount of blood transfused, the type and amount of antibody involved in the reaction, and whether the recipient has had previous sensitization.

Monitoring the patient carefully during the transfusion period is essential in recognizing early signs of a transfusion reaction, including those that may become life threatening. A general guideline for patient monitoring is first to start the transfusion slowly during the first 15 minutes. Monitor temperature, pulse, and respiration every 15 minutes for the first hour, 1 hour after the end of the transfusion, and every 12 hours minimally thereafter. Also obtain a PCV immediately before the transfusion, 1 hour after the transfusion has been stopped, and every 12 hours thereafter. Monitor coagulation parameters such as an ACT and platelet count at least daily in patients requiring transfusion therapy.

The most common documented clinical signs of a transfusion reaction include pyrexia, urticaria, salivation/ptyalism, nausea, chills, and vomiting. Other clinical signs of a transfusion reaction may include tachycardia, tremors, collapse, dyspnea, weakness, hypotension, collapse, and seizures. Severe intravascular hemolytic reactions may occur within minutes of the start of the transfusion, causing hemoglobinemia, hemoglobinuria, disseminated intravascular coagulation, and clinical signs of shock. Extravascular hemolytic reactions typically occur later and will result in hyperbilirubinemia and bilirubinuria.

Pretreatment of patients to help decrease the risk of a transfusion reaction remains controversial, and in most cases, pretreatment with glucocorticoids and antihistamines is ineffective at preventing intravascular hemolysis and other reactions should they occur. The most important component of preventing a transfusion reaction is to screen each recipient carefully and process the donor component therapy carefully before the administration of any blood products. Treatment of a transfusion reaction depends on its severity. In all cases, stop the transfusion immediately when clinical signs of a reaction occur. In most cases, discontinuation of the transfusion and administration of drugs to stop the hypersensitivity reaction will be sufficient. Once the medications have taken effect, restart the transfusion slowly and monitor the patient carefully for further signs of reaction. In more severe cases in which a patient's cardiovascular or respiratory system become compromised and hypotension, tachycardia, or tachypnea occurs, immediately discontinue the transfusion and administer diphenhydramine (1 mg/kg IM), dexamethasone-sodium phosphate (0.25 to 0.5 mg/kg IV), and epinephrine to the patient. The patient should have a urinary catheter and central venous catheter placed for measurement of urine output and central venous pressures. Aggressive fluid therapy may be necessary to avoid renal insufficiency or renal damage associated with severe intravascular hemolysis. Overhydration with subsequent pulmonary edema generally can be managed with supplemental oxygen administration and intravenous or intramuscular administration of furosemide (2 to 4 mg/kg). Plasma products with or without heparin can be administered for disseminated intravascular coagulation.

Hemoglobin-based oxygen carriers

Hemoglobin-based oxygen carriers (HBOCs) are currently in the forefront of veterinary and human transfusion medicine. Stroma-free purified bovine hemoglobin is currently available for use in dogs and cats when fresh or stored red blood products are unavailable.

The HBOCs can be stored at room temperature and have a relatively long shelf life compared with red blood component products. The HBOCs function to carry oxygen through the blood and can diffuse oxygen past areas of poor tissue perfusion. An additional characteristic of HBOCs is as a potent colloid, serving to maintain fluid within the vascular space. For this reason, HBOCs must be used with caution in euvolemic patients and patients with cardiovascular insufficiency.

Additional Reading

- Day TK: Current development and use of hemoglobin based oxygen carrier solutions, *J Vet Emerg Crit Care* 13(2):77-93, 2003.
- Gibson GR, Callan MB, Hoffman V, et al: Use of a hemoglobin-based oxygen-carrying solution in cats: 72 cases (1998-2000), *J Am Vet Med Assoc* 221:96-102, 2002.
- Hale AS: Canine blood groups and their importance in veterinary transfusion medicine, *Vet Clin North Am Small Anim Pract* 25(6):1323-1332, 1995.
- Harrell KA, Kristensen AT: Canine transfusion reactions and their management, *Vet Clin North Am Small Anim Pract* 25(6):1333-1361, 1995.
- Jutkowitz LA, Rozanski EA, Moreau JA, et al: Massive transfusion in dogs: 15 cases (1997-2001), *J Am Vet Med Assoc* 220:1664-1669, 2002.
- Kirby R: Transfusion therapy in emergency and critical care medicine, *Vet Clin North Am Small Anim Pract* 25:1365-1386, 1995.
- Kristensen AT, Feldman BF: General principles of small animal blood component administration, *Vet Clin North Am Small Anim Pract* 25(6):1277-1290, 1995.
- Muir WW, Wellman ML: Hemoglobin solutions and tissue oxygenation, *J Vet Intern Med* 17(2):127-135, 2003.
- Schneider A: Blood components: collection, processing and storage, *Vet Clin North Am Small Anim Pract* 25(6):1245-1261, 1995.
- Waddell LS, Holt DE, Hughes D, et al: The effect of storage on ammonia concentrations in canine packed red blood cells, *J Vet Emerg Crit Care* 11(1):23-26, 2001.

CENTRAL VENOUS PRESSURE MEASUREMENT

Central venous pressure (CVP) measures the hydrostatic pressure in the anterior vena cava and is influenced by vascular fluid volume, vascular tone, function of the right side of the heart, and changes in intrathoracic pressure during the respiratory cycle. The CVP is not a true measure of blood volume but is used to gauge fluid therapy as a method of determining how effectively the heart can pump the fluid that is being delivered to it. Thus the CVP reflects the interaction of the vascular fluid volume, vascular tone, and cardiac function. Measure CVP in any patient with acute circulatory failure, large volume fluid diuresis (i.e., toxin or oliguric or anuric renal failure), fluid in-and-out monitoring, and cardiac dysfunction. The placement of central venous catheters and thus CVP measurements is contraindicated in patients with known coagulopathies including hypercoagulable states.

To perform CVP monitoring, place a central venous catheter in the right or left jugular vein. In cats and small dogs, however, a long catheter placed in the lateral or medial saphenous vein can be used for trends in CVP monitoring. First, assemble the equipment necessary for jugular catheter (see Vascular Access Techniques for how to place a jugular or saphenous long catheter) and CVP monitoring (Box 1-7). After placing the jugular catheter, take a lateral thoracic radiograph to ensure that the tip of the catheter sits just outside of the right atrium for proper CVP measurements (see Figure 1-13).

BOX 1-7 SUPPLIES NEEDED FOR CENTRAL VENOUS PRESSURE MONITORING

- Two lengths of intravenous extension tubing
- Three-way stopcock
- Heparinized 0.9% saline solution
- 20-mL syringe
- Manometer or ruler (centimeter)

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To establish an intravenous catheter for CVP, follow this procedure:

1. Assemble the CVP setup such that the male end of a length of sterile intravenous catheter extension tubing is inserted into the T port of the jugular or medial/lateral saphenous catheter. Make sure to flush the length of tubing with sterile saline before connecting it to the patient to avoid iatrogenic air embolism.
2. Next, insert the male end of a three-way stopcock into the female end of the extension tubing.
3. Attach a 20-mL syringe filled with heparinized sterile 0.9% saline to one of the female ports of the three-way stopcock and either a manometer or a second length of intravenous extension tubing attached to a metric ruler.
4. Lay the patient in lateral or sternal recumbancy.
5. Turn the stopcock OFF to the manometer/ruler and ON to the patient. Infuse a small amount of heparinized saline through the catheter to flush the catheter.
6. Next, turn the stopcock OFF to the patient and ON to the manometer. Gently flush the manometer or length of extension tubing with heparinized saline from the syringe. Use care not to agitate the fluid and create air bubbles within the line or manometer that will artifactually change the CVP measured.
7. Next, lower the 0 cm point on the manometer or ruler to the level of the patient's manubrium (if the patient is in lateral recumbancy) or the point of the elbow (if the patient is in sternal recumbancy).
8. Turn the stopcock OFF to the syringe, and allow the fluid column to equilibrate with the patient's intravascular volume. Once the fluid column stops falling and the level rises and falls with the patient's heartbeat, measure the number adjacent to the bottom of the meniscus of the fluid column. This is the CVP in centimeters of water (see Figure 1-4).
9. Repeat the measurement several times with the patient in the same position to make sure that none of the values has been increased or decreased artifactually in error. Alternately, attach the central catheter to a pressure transducer and perform electronic monitoring of CVP.

There is no absolute value for normal CVP. The normal CVP for small animal patients is 0 to 5 cm H₂O. Values less than zero are associated with absolute or relative hypovolemia. Values of 5 to 10 cm H₂O are borderline hypervolemia, and values greater than 10 cm H₂O suggest intravascular volume overload. Values greater than 15 cm H₂O may be correlated with congestive heart failure and the development of pulmonary edema. In individual patients, the trend in change in CVP is more important than absolute values. As a rule of thumb, when using CVP measurements to gauge fluid therapy and avoid vascular and pulmonary overload, the CVP should not increase by more than 5 cm H₂O in any 24-hour period. If an abrupt increase in CVP is found, repeat the measurement to make sure that the elevated value was not obtained in error. If the value truly has increased dramatically, temporarily discontinue fluid therapy and consider administration of a diuretic.

Additional Reading

- DeLaforcade AM, Rozanski EA: Central venous pressure and arterial blood pressure measurements, *Vet Clin North Am Small Anim Pract* 31(6):1163-1174, 2001.
- Gookin JL, Atkins CE: Evaluation of the effects of pleural effusion on the central venous pressure in cats, *J Vet Intern Med* 13(6):561-563, 1999.
- Oakley RE, Olivier B, Eyster GE, et al: Experimental evaluation of central venous pressure monitoring in the dog, *J Am Anim Hosp Assoc* 33:77-82, 1997.
- Waddell LS: Direct blood pressure monitoring, *Clin Tech Small Anim Pract* 15(3):111-118, 2000.

FLUID THERAPY

The diagnosis of intracellular fluid deficit is difficult and is based more on the presence of hypernatremia or hyperosmolality than on clinical signs. An intracellular fluid deficit is expected when free water loss by insensible losses and vomiting, diarrhea, or urine is not matched by free water intake. Consideration of the location of the patient's fluid deficit,

TABLE 1-5 Correlation of Clinical Signs with Estimated Percent Dehydration

Clinical signs of interstitial dehydration	Percent (%) deficit
History of vomiting and diarrhea, no visible clinical signs of deficit	4%
Dry mucous membranes, mild skin tenting	5%
Increased skin tenting, dry mucous membranes, mild tachycardia, normal pulse*	7%
Increased skin tenting, dry mucous membranes, tachycardia, weak pulse pressure	10%
Increased skin tenting, dry corneas, dry mucous membranes, elevated or decreased heart rate, poor pulse quality, altered level of consciousness*	12%

*Note: These measures are largely subjective because patients with severe weight loss and loss of subcutaneous fat and very young and very old animals may have increased skin tenting or sunken eyes even in the absence of dehydration.

degree and type of acid-base and electrolyte disorders, and the presence of any ongoing fluid losses should dictate and help guide each patient's individualized fluid therapy plan (Table 1-5).

ACID-BASE PHYSIOLOGY

Normal pH in dogs and cats ranges from 7.30 to 7.45. The three major mechanisms that maintain blood pH within a normal physiologic range include buffering systems, respiratory mechanisms that alter carbon dioxide, and renal (metabolic) mechanisms that retain or excrete hydrogen and bicarbonate ions. The metabolic contribution to acid-base balance can be estimated by measuring total carbon dioxide and pH or by calculating the bicarbonate or base deficit/excess values. Hydrogen and bicarbonate ions have an important influence on normal structure and function of cellular proteins. Treat acidemia if the bicarbonate is less than 12 mEq/L, if the pH is less than 7.2, or if the base deficit is less than -10 mEq/L. Normal bicarbonate concentration is 18 to 26 mEq/L in dogs and 17 to 23 mEq/L in cats (Boxes 1-8 and 1-9).

BOX 1-8 DIFFERENTIAL DIAGNOSES FOR METABOLIC ALKALOSIS

CHLORIDE RESPONSIVE

Vomiting of stomach contents
Diuretic therapy
Posthyperventilation

CHLORIDE RESISTANT

Primary hyperaldosteronism
Hyperadrenocorticism

ALKALI ADMINISTRATION

Oral administration of sodium bicarbonate or other organic anions (e.g., lactate, citrate, gluconate, and acetate)
Oral administration of cation exchange resin with nonabsorbable alkali (e.g., phosphorus binder)

MISCELLANEOUS

Refeeding after fasting
High-dose penicillin
Severe potassium or magnesium deficiency

Modified from DiBartola SP: *Fluid, electrolyte and acid-base disorders in small animal practice*, St Louis, 2005, Saunders.

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BOX 1-9 DIFFERENTIAL DIAGNOSES FOR METABOLIC ACIDOSIS

INCREASED ANION GAP (NORMOCHLOREMIC)

Ethylene glycol intoxication
 Salicylate intoxication
 Other rare intoxications (e.g., paraldehyde or methanol)
 Diabetic ketoacidosis^{*}
 Uremic acidosis[†]
 Lactic acidosis

NORMAL ANION GAP (HYPERCHLOREMIC)

Diarrhea
 Renal tubular acidosis
 Carbonic anhydrase inhibitors (e.g., acetazolamide)
 Ammonium chloride
 Cationic amino acids (e.g., lysine, arginine, and histidine)
 Posthypocapnic metabolic acidosis
 Dilutional acidosis (e.g., rapid administration of 0.9% saline)
 Hypoadrenocorticism[‡]

Modified from DiBartola SP: *Fluid, electrolyte and acid-base disorders in small animal practice*, St Louis, 2005, Saunders.

*Patients with diabetic ketoacidosis may have some component of hyperchloremic metabolic acidosis along with increased anion gap acidosis.

†The metabolic acidosis early in renal failure may be hyperchloremic and later may convert to typical increased anion gap acidosis.

‡Patients with hypoadrenocorticism typically have hyponatremia because of impaired water excretion, absence of aldosterone, impaired renal function, and lactic acidosis. These factors prevent manifestation of hyperchloremia.

The respiratory system further contributes to acid-base status by changes in the elimination of carbon dioxide. Hyperventilation decreases the blood PCO_2 and causes a respiratory alkalosis. Hypoventilation increases the blood PCO_2 and causes a respiratory acidosis. Depending on the altitude, the PCO_2 in dogs can range from 32 to 44 mm Hg. In cats, normal is 28 to 32 mm Hg. Venous PCO_2 values are 33 to 50 mm Hg in dogs and 33 to 45 mm Hg in cats.

Use a systematic approach whenever attempting to interpret a patient's acid-base status. Ideally, obtain an arterial blood sample so that you can monitor the patient's oxygenation and ventilation. Once an arterial blood sample has been obtained, follow these steps:

1. Determine whether the blood sample is arterial or venous by looking at the oxygen saturation (SaO_2). The SaO_2 should be greater than 90% if the sample is truly arterial, although it can be as low as 80% if a patient has severe hypoxemia.
2. Consider the patient's pH. If the pH is outside of the normal range, an acid-base disturbance is present. If the pH is within the normal range, an acid-base disturbance may or may not be present. If the pH is low, the patient is acidotic. If the pH is high, the patient is alkalotic.
3. Next, look at the base excess or deficit. If the base excess is increased, the patient has higher than normal bicarbonate. If there is a base deficit, the patient may have a low bicarbonate or increase in unmeasured anions (e.g., lactic acid or ketoacids).
4. Next, look at the bicarbonate. If the pH is low AND the bicarbonate is low, the patient has a metabolic acidosis. If the pH is high AND the bicarbonate is elevated, the patient has a metabolic alkalosis.
5. Next, look at the PaCO_2 . If the patient's pH is low and the PaCO_2 is elevated, the patient has a respiratory acidosis. If the patient's pH is high and the PaCO_2 is low, the patient has a respiratory alkalosis.
6. Finally, if you are interested in the patient's oxygenation, look at the PaO_2 . Normal PaO_2 is greater than 80 mm Hg.

TABLE 1-6 Compensatory Renal and Respiratory Responses in Patients with Primary Acid-Base Disorders

Disorder	Primary change	Compensatory response
Metabolic acidosis	$\downarrow \text{HCO}_3^-$	0.7 mm Hg decrement in PCO_2 for each 1 mEq/L decrement in HCO_3^-
Metabolic alkalosis	$\uparrow \text{HCO}_3^-$	0.7 mm Hg increment in PCO_2 for each 1 mEq/L increment in HCO_3^-
Acute respiratory acidosis	$\uparrow \text{PCO}_2$	1.5 mEq/L increment in HCO_3^- for each 10 mm Hg increment in PCO_2
Chronic respiratory acidosis	$\uparrow \text{PCO}_2$	3.5 mEq/L increment in HCO_3^- for each 10 mm Hg increment in PCO_2
Acute respiratory alkalosis	$\downarrow \text{PCO}_2$	2.5 mEq/L decrement in HCO_3^- for each 10 mm Hg decrement in PCO_2
Chronic respiratory alkalosis	$\downarrow \text{PCO}_2$	5.5 mEq/L decrement in HCO_3^- for each 10 mm Hg decrement in PCO_2

From DiBartola SP: *Fluid, electrolyte and acid-base disorders in small animal practice*, St Louis, 2005, Saunders.

TABLE 1-7 Acid-Base Values in Acute Uncompensated Disturbances

Disturbance	pH	PaCO_2	Sodium bicarbonate
Metabolic acidosis	\downarrow	—	\downarrow
Metabolic alkalosis	\uparrow	—	\uparrow
Respiratory acidosis	\downarrow	\downarrow	—
Respiratory alkalosis	\uparrow	\downarrow	—

- Next, you must determine whether the disorders present are primary disorders or an expected compensation for disorders in the opposing system. For example, is the patient retaining bicarbonate (metabolic alkalosis) because of carbon dioxide retention (respiratory acidosis)? Use the chart in Table 1-6 to evaluate whether the appropriate degree of compensation is occurring. If the adaptive response falls within the expected range, a *simple* acid-base disorder is present. If the response falls outside of the expected range, a *mixed* acid-base disorder is likely present.
- Finally, you must determine whether the patient's acid-base disturbance is compatible with the history and physical examination findings. If the acid-base disturbance does not fit with the patient's history and physical examination abnormalities, question the results of the blood gas analyses and possibly repeat them.

The most desirable method of assessing the acid-base status of an animal is with a blood gas analyzer. Arterial samples are preferred over venous samples, with heparin used as an anticoagulant (Table 1-7).

ELECTROLYTE MAINTENANCE AND ABNORMALITIES

Potassium

Potassium primarily is located in the intracellular fluid compartment. Serum potassium is regulated by the actions of the sodium-potassium-adenosinetriphosphatase pump on cellular membranes, including those of the renal tubular epithelium. Inorganic metabolic acidosis artifactually can raise serum potassium levels because of redistribution of extracellular potassium in exchange for intracellular hydrogen ion movement in an attempt to correct serum pH.

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Potassium is one of the major players in the maintenance of resting membrane potentials of excitable tissue, including neurons and cardiac myocytes. Changes in serum potassium can affect cardiac conduction adversely. Hyperkalemia lowers the resting membrane potential and makes cardiac cells, particularly those of the atria, more susceptible to depolarization. Characteristic signs of severe hyperkalemia that can be observed on an ECG rhythm strip include an absence of P waves, widened QRS complexes, and tall tented or spiked T waves. Further increases in serum potassium can be associated with bradycardia, ventricular fibrillation, and cardiac asystole (death). Treatment of hyperkalemia consists of administration of insulin (0.25 to 0.5 units/kg, IV regular insulin) and dextrose (1 g dextrose per unit of insulin administered, followed by 2.5% dextrose IV CRI to prevent hypoglycemia), calcium (2 to 10 mL of 10% calcium gluconate administered IV slowly to effect), or sodium bicarbonate (1 mEq/kg, IV slowly). Insulin plus dextrose and bicarbonate therapy help drive the potassium intracellularly, whereas calcium antagonizes the effect of hyperkalemia on the myocardial cells. All of the treatments work within minutes, although the effects are relatively short-lived (20 minutes to 1 hour) unless the cause of the hyperkalemia is identified and treated appropriately (Box 1-10). Dilution of serum potassium also results from restoring intravascular fluid volume and correcting metabolic acidosis, in most cases. Treatment with a fluid that does not contain potassium (preferably 0.9% sodium chloride) is recommended.

Hypokalemia elevates the resting membrane potential and results in cellular hyperpolarization. Hypokalemia may be associated with ventricular dysrhythmias, but the ECG changes are not as characteristic as those observed with hyperkalemia. Causes of hypokalemia include renal losses, anorexia, gastrointestinal loss (vomiting, diarrhea), intravenous fluid diuresis, loop diuretics, and postobstructive diuresis (Box 1-11). If the serum potassium concentration is known, potassium supplementation in the form of potassium chloride or potassium phosphate can be added to the patient's intravenous fluids. Correct serum potassium levels less than 3.0 mEq/L or greater than 6.0 mEq/L. Potassium rates should not exceed 0.5 mEq/kg/hour (Table 1-8).

Bicarbonate concentration

Metabolic acidosis from bicarbonate depletion often corrects itself with volume restoration in most small animal patients. Patients with moderate to severe metabolic acidosis may benefit from bicarbonate supplementation therapy. The metabolic contribution to acid-base balance is identified by measuring the total carbon dioxide concentration or calculating the bicarbonate concentration. If these measurements are not available, the degree of expected metabolic acidosis can be estimated subjectively by the severity of underlying disease that often contributes to metabolic acidosis: hypovolemic or traumatic shock, septic shock, diabetic ketoacidosis, or oliguric/anuric renal failure. If the metabolic acidosis is estimated to be mild, moderate, or severe, add sodium bicarbonate at 1, 3, and 5 mEq/kg body mass, respectively. Patients with diabetic ketoacidosis may not require bicarbonate administration once volume replacement and perfusion is restored, and the ketoacids are metabolized to bicarbonate. If the bicarbonate measurement of base deficit is known, the following formula can be used as a gauge for bicarbonate supplementation:

$$\text{Base deficit} \times 0.3 = \text{Body mass (kg)} = \text{mEq Bicarbonate to administer}$$

Osmolality

Osmolality is measured by freezing point depression or a vapor pressure osmometer, or it may be calculated by the following formula:

$$\text{mOsm/kg} = 2[(\text{Na}^+) + (\text{K}^+)] + \text{BUN}/2.8 + \text{Glucose}/18$$

where sodium and potassium are measured in milliequivalents, and BUN and glucose are measured in milligrams per deciliter. Osmolalities less than 260 mOsm/kg or greater

BOX 1-10 DIFFERENTIAL DIAGNOSES FOR HYPERKALEMIA**PSEUDOHYPERKALEMIA**

Thrombocytosis
Hemolysis

INCREASED INTAKE

Unlikely to cause hyperkalemia in presence of normal renal function unless iatrogenic (e.g., continuous infusion of potassium-containing fluids at an excessively rapid rate)

TRANSLOCATION (INTRACELLULAR FLUID TRANSFER TO EXTRACELLULAR FLUID)

Acute mineral acidosis (e.g., hydrochloric acid or ammonium chloride)
Insulin deficiency (e.g., diabetic ketoacidosis)
Acute tumor lysis syndrome
Reperfusion of extremities after aortic thromboembolism in cats with cardiomyopathy
Hyperkalemic periodic paralysis (one case report in a pit bull)
Mild hyperkalemia after exercise in dogs with induced hypothyroidism
Infusion of lysine or arginine in total parenteral nutrition solutions

DRUGS

Nonspecific β -blockers (e.g., propranolol)*
Cardiac glycosides (e.g., digoxin)*

DECREASED URINARY EXCRETION

Urethral obstruction
Ruptured bladder
Anuric or oliguric renal failure
Hypoadrenocorticism
Selected gastrointestinal disease (e.g., trichuriasis, salmonellosis, or perforated duodenal ulcer)
Late pregnancy in Greyhound dogs (mechanism unknown but affected dogs had gastrointestinal fluid loss)
Chylothorax with repeated pleural fluid drainage
Hyporeninemic hypoaldosteronism†

DRUGS

Angiotensin-converting enzyme inhibitors (e.g., enalapril)*
Angiotensin receptor blockers (e.g., losartan)*
Cyclosporine and tacrolimus*
Potassium-sparing diuretics (e.g., spironolactone, amiloride, and triamterene)*
Nonsteroidal antiinflammatory drugs*
Heparin*
Trimethoprim*

From DiBartola SP: *Fluid, electrolyte and acid-base disorders in small animal practice*, St Louis, 2005, Saunders.

*Likely to cause hyperkalemia only in conjunction with other contributing factors (e.g., other drugs, decreased renal function, or concurrent administration of potassium supplements).

†Not well documented in veterinary medicine.

than 360 mOsm/kg are serious enough to warrant therapy. The difference between the measured osmolality and the calculated osmolality (the osmolal gap) should be less than 10 mOsm/kg. If the osmolal gap is greater than 20 mOsm/kg, consider the presence of unmeasured anions such as ethylene glycol metabolites.

Sodium

The volume of extracellular fluid is determined by the total body sodium content, whereas the osmolality and sodium concentration are determined by water balance. Serum sodium concentration is an indication of the amount of sodium relative to water in the extracellular fluid and provides no direct information about the total body sodium content.

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BOX 1-11 DIFFERENTIAL DIAGNOSES FOR HYPOKALEMIA

DECREASED INTAKE

Alone unlikely to cause hypokalemia unless diet is aberrant
 Administration of potassium-free (e.g., 0.9% sodium chloride or 5% dextrose in water) or potassium-deficient fluids (e.g., lactated Ringer's solution over several days)
 Bentonite clay ingestion (e.g., cat litter)

TRANSLOCATION (EXTRACELLULAR FLUID TRANSFER TO INTRACELLULAR FLUID)

Alkalemia
 Insulin/glucose-containing fluids
 Catecholamines
 Hypothermia
 Hypokalemic periodic paralysis (Burmese cats)
 Albuterol overdosage

INCREASED LOSS

Gastrointestinal (FE_K less than 4% to 6%)
 Vomiting of stomach contents
 Diarrhea
 Urinary (fractional excretion of potassium [FE_K] greater than 4% to 6%)
 Chronic renal failure in cats
 Diet-induced hypokalemic nephropathy in cats
 Distal (type I) renal tubular acidosis
 Proximal (type II) renal tubular acidosis after sodium bicarbonate treatment
 Postobstructive diuresis
 Dialysis
 Mineralocorticoid excess
 Hyperadrenocorticism
 Primary hyperaldosteronism (adenoma, adenocarcinoma, hyperplasia)

DRUGS

Loop diuretics (e.g., furosemide and ethacrynic acid)
 Thiazide diuretics (e.g., chlorothiazide and hydrochlorothiazide)
 Amphotericin B
 Penicillins
 Unknown mechanism
 Rattlesnake envenomation

From DiBartola SP: *Fluid, electrolyte and acid-base disorders in small animal practice*, St Louis, 2005, Saunders.

TABLE 1 - 8 Guidelines for Routine Intravenous Potassium Supplementation in Dogs and Cats

Serum potassium (mEq/L)	Potassium chloride to add to 250 mL of fluid (mEq)	Potassium chloride to add to 1 L of fluid (mEq)	Maximal fluid rate (mL/kg/hour)*
<2.0†	20	80	6
2.1-2.5	15	60	8
2.6-3.0	10	40	12
3.1-3.5	7	28	18
3.6-5.0	5	20	25

*Maximal rate of potassium supplementation should not exceed 0.5 mEq/kg/hour.

†If refractory hypokalemia is present, supplement magnesium at 0.75 mEq/kg/day for 24 hours.

Patients with hyponatremia or hypernatremia may have decreased, normal, or increased total body sodium content (Boxes 1-12 and 1-13). An increased serum sodium concentration implies hyperosmolality, whereas a decrease in serum sodium concentration usually, but not always, implies hypoosmolality. The severity of clinical signs of hypernatremia and hyponatremia is related primarily to the rapidity of the onset of the change rather than to the magnitude of the associated plasma hyperosmolality or hypoosmolality. Clinical signs of neurologic disturbances include disorientation, ataxia, and seizures, and coma may occur at serum sodium concentrations less than 120 mEq/L or greater than 170 mEq/L in dogs.

BOX 1-12 DIFFERENTIAL DIAGNOSES FOR HYPONATREMIA

WITH NORMAL PLASMA OSMOLALITY

Hyperlipemia
Hyperproteinemia

Antidiuretic drugs
Myxedema coma of hypothyroidism
Hypotonic fluid infusion
And hypovolemia

WITH HIGH PLASMA OSMOLALITY

Hyperglycemia
Mannitol infusion

Gastrointestinal loss
Vomiting
Diarrhea

WITH LOW PLASMA OSMOLALITY

And hypervolemia
Severe liver disease
Congestive heart failure
Nephrotic syndrome
Advanced renal failure
And normovolemia
Psychogenic polydipsia
Syndrome of inappropriate antidiuretic hormone secretion

Third-space loss
Pancreatitis
Peritonitis
Uroabdomen
Pleural effusion (e.g., chylothorax)
Peritoneal effusion
Cutaneous loss
Burns
Hypoadrenocorticism
Diuretic administration

From DiBartola SP: *Fluid, electrolyte and acid-base disorders in small animal practice*, St Louis, 2005, Saunders.

BOX 1-13 DIFFERENTIAL DIAGNOSES FOR HYPERNATREMIA

PURE WATER DEFICIT

Primary hypodipsia (e.g., in Miniature schnauzers)
Diabetes insipidus
Central
Nephrogenic
High environmental temperature
Fever
Inadequate access to water

Renal

Osmotic diuresis
Diabetes mellitus
Mannitol infusion
Chemical diuretics
Chronic renal failure
Nonoliguric acute renal failure
Postobstructive diuresis

HYPOTONIC FLUID LOSS

Extrarenal
Gastrointestinal
Vomiting
Diarrhea
Small intestinal obstruction
Third-space loss
Peritonitis
Pancreatitis
Cutaneous
Burns

IMPERMEANT SOLUTE GAIN

Salt poisoning
Hypertonic fluid administration
Hypertonic saline
Sodium bicarbonate
Parenteral nutrition
Sodium phosphate enema
Hyperaldosteronism
Hyperadrenocorticism

From DiBartola SP: *Fluid, electrolyte and acid-base disorders in small animal practice*, St Louis, 2005, Saunders.

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Therapy of hypernatremia or hyponatremia with fluid containing low or higher concentrations of sodium should proceed with caution, for rapid changes (decreases or increases) of serum sodium and osmolality can cause rapid changes in the intracellular and extracellular fluid flux, leading to intracellular dehydration or edema, even though the serum sodium has not been returned to normal. A rule of thumb is to not raise or lower the serum sodium by more than 15 mEq/L during any one 24-hour period. Restoration of the serum sodium concentration over a period of 48 to 72 hours is better. In almost all circumstances, an animal will correct its sodium balance with simple fluid restoration. If severe hypernatremia exists that suggests a free water deficit, however, the free water deficit should be calculated from the following formula:

$$\text{Free water deficit} = 0.4 \times \text{Body mass (kg)} \times \{[(\text{Plasma sodium})/140] - 1\}$$

Hypernatremia can be corrected slowly with 0.45% sodium chloride plus 2.5% dextrose, 5% dextrose in water, or lactated Ringer's solution (sodium content: 130 mEq/L). Correct hyponatremia initially with 0.9% sodium chloride.

Anion gap

Sodium is balanced predominantly by chloride and bicarbonate. The difference between these concentrations, $(\text{Na}^+) - [(\text{Cl}^-) + (\text{HCO}_3^-)]$, has been called the *anion gap*. The normal anion gap is between 12 and 25 mEq/L. When the anion gap exceeds 25, consider the possibility of an accumulation of unmeasured anions (e.g., lactate, ketoacids, phosphate, sulfate, ethylene glycol metabolites, and salicylate). Abnormalities in the anion gap may be helpful in determining the cause of metabolic acidosis (Boxes 1-14 and 1-15).

ONCOTIC PRESSURE

The colloid oncotic pressure of blood is associated primarily with large-molecular-weight colloidal substances in circulation. The major player in maintaining intravascular and interstitial oncotic pressure, the water-retaining property of each fluid compartment, is albumin. Albumin contributes roughly 80% to the colloidal oncotic pressure of blood. The majority of albumin is located within the interstitial space. Hypoalbuminemia can result from increased loss in the form of protein-losing enteropathy or nephropathy and wound exudates, or it may be due to lack of hepatic albumin synthesis. Serum albumin pools are

BOX 1-14 DECREASED ANION GAP

- Myeloma (immunoglobulin G)
- Hypoalbuminemia
- Dilutional from crystalloid fluid administration
- Potassium bromide therapy
- Pseudohyponatremia

BOX 1-15 INCREASED ANION GAP

- Follow the mnemonic A MUD PILE:
- Aspirin (salicylates)
- Methanol
- Uremia
- Diabetic ketoacidosis
- Phosphate, Paraldehyde
- Indomethacin
- Lactic acidosis
- Ethylene glycol intoxication

in a constant flux with interstitial albumin. Once interstitial albumin pools become depleted from replenishing serum albumin, serum albumin levels can continue to decrease, which can lead to a decrease in colloidal oncotic pressure. Serum albumin less than 2.0 g/dL has been associated with inadequate intravascular fluid retention and the development of peripheral edema and third spacing of fluid. Oncotic pressure can be restored with the use of artificial or synthetic colloids or natural colloids (see Colloids).

MAINTENANCE FLUID REQUIREMENTS

Maintenance fluid requirements have been extrapolated from the formulas used to calculate a patient's daily metabolic energy requirements because it takes 1 mL of water to metabolize 1 Kcal of energy (Table 1-9). The patient's daily metabolic water (fluid) requirements can be calculated by the following formula:

$$\text{Fluid (mL)} = [30 \times \text{Body mass (kg)}] + 70$$

Administration of an isotonic crystalloid fluid for maintenance requirements often can produce iatrogenic hypokalemia. In most cases, supplemental potassium must be added to prevent hypokalemia resulting from inappetance, kaliuresis, and supplementation with isotonic crystalloid fluids.

CALCULATION OF FLUID DEFICITS AND ONGOING LOSSES

The most reliable method of determining the degree of fluid deficit is by weighing the animal and calculating acute weight loss. Acute weight loss in a patient with volume loss in the form of vomiting, feces, wound exudates, and urine is due to fluid loss and not loss of muscle or fat. Lean body mass normally is not gained or lost rapidly enough to cause major changes in body weight. One milliliter of water weighs approximately 1 g. This fact allows calculation of the patient's fluid deficit, if ongoing losses can be measured. When a patient first presents, however, the body weight before a fluid deficit has occurred rarely is known. Instead, one must rely on subjective measures of dehydration to estimate the patient's percent dehydration and to calculate the volume of fluid required to rehydrate the patient over the next 24 hours. To calculate the volume deficit, use the following formula:

$$\text{Body mass (kg)} \times (\% \text{ dehydration}) \times 1000 = \text{Fluid deficit (mL)}$$

The patient's fluid deficit must be added to the daily maintenance fluid requirements and administered over a 24-hour period. Ongoing losses can be determined by measuring urine output, weighing the patient at least 2 to 3 times a day, and measuring the volume or weight of vomitus or diarrhea.

CRYSTALLOID AND COLLOID FLUIDS

Crystalloid fluids

A crystalloid fluid contains crystals of salts with a composition similar to that of the extracellular fluid space and can be used to maintain daily fluid requirements and replace fluid deficits or ongoing fluid losses (Table 1-10). Metabolic, acid-base, and electrolyte imbalances also can be treated with isotonic fluids with or without supplemental electrolytes and buffers. Depending on the patient's clinical condition, choose the specific isotonic crystalloid fluid to replace and maintain the patient's acid-base and electrolyte status (Table 1-11). Crystalloid fluids are readily available, are relatively inexpensive, and can be administered safely in large volumes to patients with no preexisting cardiac or renal disease or cerebral edema. Following infusion, approximately 80% of the volume of a crystalloid fluid infused will redistribute to the interstitial fluid compartment. As such, crystalloid fluids alone are ineffective for ongoing intravascular volume depletion when given as a bolus. The crystalloid fluid bolus must be followed by a constant rate infusion, taking into consideration the patient's daily maintenance fluid requirements and ongoing fluid losses. Administration of a large volume of crystalloid fluids can cause dilutional anemia and coagulopathies.

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TABLE 1 - 9 Approximate Daily Energy and Water Requirements of Dogs and Cats Based on Body Mass*		
Body mass (kg)	Total energy (Kcal) per day	Total water (mL) per hour
1	100	4.2
2	130	5.4
3	160	6.7
4	190	7.9
5	220	9.2
6	250	10.4
7	280	11.7
8	310	12.9
9	340	14.2
10	370	15.4
11	400	16.7
12	430	17.9
13	460	19.2
14	490	20.4
15	520	21.7
16	550	22.9
17	580	24.2
18	610	25.4
19	640	26.7
20	670	27.9
21	700	29.2
22	730	30.4
23	760	31.7
24	790	32.9
25	820	34.2
26	850	35.4
27	880	36.7
28	910	37.9
29	940	39.2
30	970	40.4
35	1120	46.7
40	1270	52.9
45	1420	59.2
50	1570	65.4
55	1720	71.7
60	1870	77.9
65	2020	84.2
70	2170	90.4
75	2320	96.7
80	2470	102.9
85	2620	109.2
90	2770	115.4
95	2920	121.7
100	3070	127.9

* $30 \times BW_{\text{kg}} + 70 = \text{Kcal/day} = \text{mL/day}$. Note: This formula will slightly underestimate the requirements for patients that are less than 2 kg and will slightly overestimate the requirements for patients greater than 70 kg.

TABLE 1 - 10 Electrolyte Composition (mEq/L) of Commonly Used Isotonic and Hypotonic Crystalloid Fluids

	0.9% Saline	0.45% NaCl	Lactated Ringer's	Normosol-R
Sodium	154	77	130	140
Chloride	154	77	109	98
Potassium	0	0	4	5
Calcium	0	0	3	0
Magnesium	0	0	0	3
pH	7.386	5.7	6.7	7.4
Buffer	None	None	Lactate 28	Acetate 27 Gluconate 23

TABLE 1 - 11 Indications for Use of Specific Crystalloid Fluids in Specific Disease Processes

Crystalloid fluid	Indications
Lactated Ringer's solution	Hypocalcemia, to replace dehydration deficit, metabolic acidosis, use as a maintenance fluid, renal failure
Plasmalyte-M and Normosol-R*	To replace dehydration deficit, metabolic acidosis, hypomagnesemia Use as a maintenance fluid, renal failure
0.45% sodium chloride + 2.5% dextrose	Cardiac disease, hepatic failure, hypernatremia
5% dextrose in water	Cardiac disease, hepatic failure, hypernatremia
0.9% sodium chloride	Conditions associated with hypercalcemia and hyperkalemia (e.g., hypoadrenocorticism, vitamin D toxicity, renal failure, and various neoplasias)

*Abbott Laboratories, Abbott Park, Illinois.

Obtain the patient's hematocrit before fluid infusion and regularly during the course of fluid therapy, particularly in patients with preexisting anemia or hypoproteinemia.

Colloids

A colloid is a large-molecular-weight particle that acts as an effective volume expander by drawing fluid from the interstitial fluid compartment into the intravascular space. When administered with a crystalloid, a colloid serves to hold or retain the crystalloid fluid within the vascular space for a longer time than if the crystalloid fluid were administered alone. Because of this property, colloids can promote better tissue perfusion at lower infusion volumes and equivalent colloid oncotic pressures and mean blood pressures than crystalloids. Administer the synthetic colloids in incremental boluses of 5 to 10 mL/kg over 5 to 15 minutes during the treatment of hypotension. When synthetic colloids are administered for maintenance of colloidal oncotic pressure in hypoalbuminemic/hypoproteinemic patients, the recommended dose is 20 to 30 mL/kg/day as a constant rate infusion. Because colloids

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retain fluid in the vascular space, the volume of crystalloid fluid infused (maintenance + deficit + ongoing losses) should be decreased by 25% to 50% to avoid vascular volume overload.

Two major classes of colloids exist: natural and synthetic. Natural colloids (whole blood, packed RBCs, plasma) are discussed elsewhere in this text. Concentrated human albumin is a natural purified colloid that recently has become more popular in the treatment of advanced hypoalbuminemia and hypoproteinemia and will be discussed here. Synthetic colloids are starch polymers and include dextrans and hetastarch.

Concentrated human albumin is available as a 5% or 25% solution. The 5% solution has an osmolality similar to that of serum (308 mOsm/L), whereas the 25% solution is hyperoncotic (1500 mOsm/L). A 25% albumin solution draws fluid from the interstitial space into the intravascular space. Concentrated albumin solutions often are used to restore circulating volume when synthetic colloids are not available. Albumin not only is important at maintaining the colloidal oncotic pressure of blood but also serves as a valuable free-radical scavenger and carrier of drugs and hormones necessary for normal tissue function and healing. Albumin levels less than 2.0 g/dL have been associated with increased morbidity and mortality. Concentrated human albumin solutions can be administered as an effective method of restoring interstitial and serum albumin concentrations in situations of acute and chronic hypoalbuminemia. Albumin (25%) is available in 50- and 100-mL vials and is more cost-efficient as an albumin replacement than procurement and administration of fresh frozen plasma. Recommended albumin infusion rates are 2 to 5 mL/kg over 4 hours, after pretreatment with diphenhydramine. Although concentrated human albumin is structurally similar to canine albumin, closely monitor the patient for signs of allergic reaction during and after the infusion.

Dextran-70 is a synthetic high-molecular-weight polysaccharide (sucrose polymer) with a molecular weight of 70,000 D. Particles less than 50,000 D, are cleared rapidly by the kidneys, whereas larger particles are cleared more slowly by the hepatic reticuloendothelial system. Dextran-70 can coat platelets and inhibit platelet function and so must be used with caution in patients with known coagulopathies. The total daily dosage should not exceed 40 mL/kg/day.

Hetastarch (hydroxyethyl starch) is a large-molecular-weight amylopectin polymer, has molecules with a molecular weight that exceeds 100,000 D, and has an average half-life of 24 to 36 hours in circulation. Hetastarch can bind with vWF and cause prolongation of the ACT and APTT; however, it does not cause a coagulopathy. Recommended rates of hetastarch infusion are 5- to 10-mL incremental boluses for the treatment of hypotension and 20 to 30 mL/kg/day as a constant rate infusion for maintenance of colloidal oncotic pressure.

IMPLEMENTING THE FLUID THERAPY PLAN

Many are the acceptable ways to administer the fluids prescribed for each patient based on the degree of dehydration, estimation of ongoing losses, ability to tolerate oral fluid, and metabolic, acid-base, and electrolyte derangements. Administer the fluids in a manner that is best for the patient and most appropriate for the practice.

To determine the rate of intravenous fluid infusion, take the total volume of fluids that have been prescribed and divide the total volume by the total number of hours in a day that intravenous fluids can be delivered safely and monitored. The safest and most accurate way to deliver intravenous fluids, particularly in extremely small animals or those with congestive heart failure, is through an intravenous fluid pump. Fluid should not be administered intravenously if the patient cannot be monitored to make sure that the fluids are being delivered at a safe rate and that the fluid line has not become disconnected.

Supplement fluids over as many hours as possible to allow the patient as much time as possible to redistribute and fully utilize the fluids administered. Fluids administered too quickly can cause a diuresis to occur, such that the majority of the fluids administered will be excreted in the urine. If time is limited or if extra time is needed for safe administration of fluids, consider using a combination of intravenously and subcutaneously

administered fluids. Intravenous is the preferred route of administration of fluids in any patient with dehydration and hypovolemia. As intravascular volume depletion occurs, reflex peripheral vasoconstriction occurs to restore core perfusion. The subcutaneous tissue are not perfused well and therefore fluids administered subcutaneously will not be absorbed well into the interstitial and intravascular spaces. Subcutaneously administered fluids can be absorbed slowly and delivered effectively in the management of mild interstitial dehydration and in the treatment of renal insufficiency. Subcutaneously administered fluids should never take the place of intravenously administered fluids in a hypovolemic patient or one with severe interstitial dehydration.

Intramedullary (intraosseous) infusion works well in small patients in which vascular access cannot be established. Shock doses of fluids and other substances, including blood products, can be administered under pressure through an intraosseous cannula. Because of the inherent discomfort and risk of osteomyelitis with intraosseous infusion, establish vascular access as soon as possible.

RATES OF ADMINISTRATION

The safest and most efficient method of intravenous fluid infusion is through a fluid pump. In cases in which a fluid pump is unavailable, infusion by gravity feed is the next option. Infusion sets from various manufacturers have calibrated drip chambers such that a specific number of drops will equal 1 mL of fluid. Fluid rates can be calculated based on the number of drops that fall into the drip chamber per minute:

$$\frac{\text{Fluid volume to be infused (mL)}}{\text{Number of hours available}} = \text{mL/hour}$$

Many pediatric drip sets deliver 60 drops/mL, such that milliliters/hour equals drops/minute. Carefully record fluid orders so that the volume to be administered is recorded as milliliters/hour, milliliters/day, and drops/minute. This will allow personnel to detect major discrepancies and calculation errors more readily. The volume actually delivered should be recorded in the record by nursing personnel. All additives should be listed clearly on the bottle on a piece of adhesive tape or a special label manufactured for this purpose. A strip of adhesive tape also can be attached to the bottle and marked appropriately to provide a quick visualization of the estimate of volume delivered.

Additional Reading

- Greco DS: The distribution of body water and general approach to the patient, *Vet Clin North Am* 28(3):473-482, 1998.
- Kirby R, Rudloff E: The critical need for colloids: maintaining fluid balance, *Compend Contin Educ Pract Vet* 19(6):705-716, 1997.
- Mathews KA: The various types of parenteral fluids and their indications, *Vet Clin North Am Small Anim Pract* 28(3):483-513, 1998.
- Mazzaferro EM, Rudloff E, Kirby R: The role of albumin in health and disease, *J Vet Emerg Crit Care* 12(2):113-124, 2002.
- Moore LE: Fluid therapy in the hypoproteinemic patient, *Vet Clin North Am Small Anim Pract* 28(3):709-715, 1998.
- Otto CM, McCall-Kauffman G, Crowe DT: Intraosseous infusion of fluids and therapeutics, *Compend Contin Educ Pract Vet* 11:421-430, 1989.
- Rozanski E, Rondeau M: Choosing fluids in traumatic hypovolemic shock: the role of crystalloids, colloids and hypertonic saline, *J Am Anim Hosp Assoc* 38(6):499-501, 2002.
- Rudloff E, Kirby R: Colloid and crystalloid resuscitation, *Vet Clin North Am Small Anim Pract* 31(6):1207-1229, 2001.

OROGASTRIC LAVAGE

Orogastric lavage is indicated for gastric decontamination of most types of toxins, for elimination of food during food bloat, and for gastric decompression during surgery for gastric dilatation-volvulus syndrome (GDV). Equipment needed to perform an orogastric lavage

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includes a large-bore flexible orogastric lavage tube, permanent marker or white tape, lubricating jelly, warm water, two large buckets, a roll of 2-inch white tape, and a manual lavage pump.

- To perform the orogastric lavage, follow this procedure:
1. Place all animals under general anesthesia with a cuffed endotracheal tube in place to protect the airway and prevent aspiration of gastric contents into the lungs.
 2. Place a roll of 2-inch white tape into the animal's mouth, and secure the tape around the muzzle. You will insert the tube through the hole in the center of the roll of tape.
 3. Next, place the distal end of the tube at the level of the last rib, directly adjacent to the animal's thorax and abdomen. Measure the length of the tube from the most distal end to the point where it comes out of the mouth, and label this location on the tube with a permanent marker or piece of white tape.
 4. Lubricate the distal portion of the tube, and gently insert it through the roll of tape in the animal's mouth.
 5. Gently push the tube down the esophagus. Palpate the tube within the esophagus. Two tubes should be palpable, the orogastric tube, and the patient's trachea. Push the tube down into the stomach. You can verify location by blowing into the proximal end of the tube and simultaneously auscultating the stomach for borborygmi.
 6. Insert the manual pump to the proximal end of the tube, and instill the warm water. Alternate instilling water with removal of fluid and gastric debris by gravity. Repeat the process until the efflux fluid is clear of any debris.
 7. Save fluid from the gastric efflux fluid for toxicologic analyses.

Additional Reading

Hackett TB: Emergency approach to intoxications, *Clin Tech Small Anim Pract* 15(2):82-87, 2000.

OXYGEN SUPPLEMENTATION

Hypoxia, or inadequate tissue oxygenation, is the primary reason for supplemental oxygen therapy. Major causes of hypoxia include hypoventilation, ventilation-perfusion mismatch, physiologic or right-to-left cardiac shunt, diffusion impairment, and decreased fraction of inspired oxygen (Table 1-12). Inadequate tissue perfusion caused by low cardiac output or vascular obstruction also can result in circulatory hypoxia. Finally, histiocytic hypoxia results from inability of cells to use oxygen that is delivered to them. This form of hypoxia can be observed with various toxin ingestions (bromethalin, cyanide) and in septic shock.

TABLE 1 - 12 Types of Hypoxia and Response to Oxygen Supplementation		
Type of hypoxia	Cause	Response to oxygen
Hypoxic hypoxia		
Alveolar hypoventilation	Central nervous system disease, drugs, rib fractures, thoracic cage damage, pneumothorax, pleural effusion	Responsive
Arteriovenous (physiologic) shunt	Pneumonia, atelectasis	Partially responsive
Diffusion impairment	Pneumonia, pulmonary edema, fibrosis, emphysema	Responsive
Decreased FIO ₂	Smoke inhalation, altitude	Responsive
Histiocytic hypoxia	Septic shock, toxins	Not very responsive
Circulatory hypoxia	Low cardiac output, vascular obstruction	Responsive

A patient's oxygenation status can be monitored invasively by drawing of arterial blood gas samples or noninvasively through pulse oximetry, in most cases (see Acid-Base Physiology and Pulse Oximetry). Inspired air at sea level has a PO_2 of 150 mm Hg. As the air travels through the upper respiratory system to the level of the alveolus, the PO_2 drops to 100 mm Hg. Tissue oxygen saturation in a normal healthy animal is 95 mm Hg. After oxygen has been delivered to the tissues, the oxygen left in the venous system (PvO_2) is approximately 40 mm Hg.

Normally, oxygen diffuses across the alveolar capillary membrane and binds reversibly with hemoglobin in RBCs. A small amount of oxygen is carried in an unbound diffusible form in the plasma. When an animal has an adequate amount of hemoglobin and hemoglobin becomes fully saturated while breathing room air, supplemental oxygen administration will only increase the SAO_2 a small amount. The unbound form of oxygen dissolved in plasma will increase. If, however, inadequate hemoglobin saturation is obtained by breathing room air, as in a case of pneumonia or pulmonary edema, for example, breathing a higher fraction of inspired oxygen (FiO_2) will improve bound and unbound hemoglobin levels. The formula for calculating oxygen content of arterial blood is as follows:

$$CaO_2 = (1.34 \times [Hb] \times SaO_2) + (0.003 \times PaO_2)$$

where CaO_2 is the arterial oxygen content, 1.34 is the amount of oxygen that can be carried by hemoglobin (Hb), SAO_2 is the hemoglobin saturation, and $0.003 \times PaO_2$ is the amount of oxygen dissolved (unbound) in plasma.

Dissolved oxygen actually contributes little to the total amount of oxygen carried in the arterial blood, and the majority depends on the amount or availability of hemoglobin and the ability of the body (pH and respiratory status) to saturate the hemoglobin at the level of the alveoli.

INDICATIONS FOR OXYGEN THERAPY

Oxygen therapy is indicated whenever hypoxia is present. The underlying cause of the hypoxia also must be identified and treated, for chronic, lifelong oxygen therapy is rarely feasible in veterinary patients. If hemoglobin levels are low due to anemia, oxygen supplementation must occur along with RBC transfusions to increase hemoglobin mass. Whenever possible, use arterial blood gas analyses or pulse oximetry to gauge a patient's response to oxygen therapy and to determine when an animal can be weaned from supplemental oxygen.

The goal of oxygen therapy is to increase the amount of oxygen bound to hemoglobin in arterial blood. Oxygen supplementation can be by hood, oxygen cage or tent, nasal or nasopharyngeal catheter, or tracheal tube. In rare cases, administration of oxygen with mechanical ventilation may be indicated.

Administration of supplemental oxygen to patients with chronic hypoxia is sometimes necessary but also dangerous. With chronic hypoxia the patient develops a chronic respiratory acidosis (elevated $Paco_2$) and depends almost entirely on the hypoxic ventilatory drive to breathe. Administration of supplemental oxygen increases PaO_2 and may inhibit the central respiratory drive, leading to hypoventilation and possibly respiratory arrest. Therefore, closely monitor animals with chronic hypoxia that are treated with supplemental oxygen.

OXYGEN HOOD

Oxygen hoods can be purchased from commercial sources or can be manufactured in the hospital using a rigid Elizabethan collar, tape, and plastic wrap. To make an oxygen hood, place several lengths of plastic wrap over the front of the Elizabethan collar and tape them in place. Leave the ventral third of the collar open to allow moisture and heat to dissipate and carbon dioxide to be eliminated. Place a length of flexible oxygen tubing under the patient's collar into the front of the hood, and run humidified oxygen at a rate of 50 to 100 mL/kg/minute. Animals may become overheated with an oxygen hood in place.

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Carefully monitor the patient's temperature so that iatrogenic hyperthermia does not occur.

OXYGEN CAGE

Commercially available plexiglass oxygen cages can be purchased from a variety of manufacturers. The best units include a mechanical thermostatically controlled compressor cooling unit, a circulatory fan, nebulizers or humidifiers to moisten the air, and a carbon dioxide absorber. Alternately, a pediatric (infant) incubator can be purchased from hospital supply sources, and humidified oxygen can be run into the cage at 2 to 10 L/minute (depending on the size of the cage). High flow rates may be required to eliminate nitrogen and carbon dioxide from the cage. In most cases, the FiO_2 inside the cage reaches 40% to 50% using this technique. Disadvantages of using an oxygen cage are high consumption/use of oxygen, rapid decrease in the FiO_2 within the cage whenever the cage must be opened for patient treatments, lack of immediate access to the patient, and potential for iatrogenic hyperthermia.

NASAL OR NASOPHARYNGEAL OXYGEN

One of the most common methods for oxygen supplementation in dogs is nasal or nasopharyngeal oxygen catheters:

1. To place a nasal or nasopharyngeal catheter, obtain a red rubber catheter (8F to 12F, depending on the size of the patient).
 - a. For nasal oxygen supplementation, measure the distal tip of the catheter from the medial canthus of the eye to the tip of the nose.
 - b. For nasopharyngeal oxygen supplementation, measure the catheter from the ramus of the mandible to the tip of the nose.
2. Mark the tube length at the tip of the nose with a permanent marker.
3. Instill topical anesthetic such as proparacaine (0.5%) or lidocaine (2%) into the nostril before placement.
4. Place a stay suture adjacent to (lateral aspect) the nostril while the topical anesthetic is taking effect.
5. Lubricate the tip of the tube with sterile lubricant.
6. Gently insert the tube into the ventral medial aspect of the nostril to the level made with the permanent marker. If you are inserting the tube into the nasopharynx, push the nasal meatus dorsally while simultaneously pushing the lateral aspect of the nostril medially to direct the tube into the ventral nasal meatus and avoid the cribriform plate.
7. Once the tube has been inserted to the appropriate length, hold the tube in place with your fingers adjacent to the nostril, and suture the tube to the stay suture. If the tube is removed, you can cut the suture around the tube and leave the stay suture in place for later use, if necessary.
8. Suture or staple the rest of the tube dorsally over the nose and in between the eyes to the top of the head, or laterally along the zygomatic arch.
9. Attach the tube to a length of flexible oxygen tubing, and provide humidified oxygen at 50 to 100 mL/kg/minute.
10. Secure an Elizabethan collar around the patient's head to prevent the patient from scratching at the tube and removing it.

MECHANICAL VENTILATION

The Rule of 60s states that if a patient's PaO_2 is less than 60 mm Hg, or if the PaCO_2 is 60 mm Hg, mechanical ventilation should be considered. For mechanical ventilation, anesthetize the patient and intubate the patient with an endotracheal tube. Alternately, a temporary tracheostomy can be performed and the patient can be maintained on a plane of light to heavy sedation and ventilated through the tracheostomy site. This method,

although technically more invasive initially, allows the patient to be awake despite requiring mechanical ventilation.

Additional Reading

- Camp-Palau MA, Marks SL, Cornick JL: Small animal oxygen therapy, *Compend Contin Educ Pract Vet* 21(7):587-597, 1999.
- Drobatz K, Hackner S, Powell S: Oxygen supplementation. In Bonagura JD, editor: *Current veterinary therapy XII. Small animal practice*, Philadelphia, 1995, WB Saunders.
- Dunphy EA, Mann FA, Dodam JR, et al: Comparison of unilateral versus bilateral nasal catheters for oxygen administration in dogs, *J Vet Emerg Crit Care* 12(4):245-251, 2002.
- Marks SL: Nasal oxygen insufflation, *J Am Anim Hosp Assoc* 35(5):366-367, 1999.

PULSE OXIMETRY

A noninvasive means of determining oxygenation is through the use of pulse oximetry. A pulse oximeter uses different wavelengths of light to distinguish characteristic differences in the properties of the different molecules in a fluid or gas mixture, in this case, oxygenated (oxyhemoglobin) and deoxygenated hemoglobin (deoxyhemoglobin) in pulsatile blood. The process is termed *pulse oximetry*.

Oxyhemoglobin and deoxyhemoglobin are different molecules that absorb and reflect different wavelengths of light. Oxyhemoglobin absorbs light in the infrared spectrum, allowing wavelengths of light in the red spectrum to transmit through it. Conversely, deoxyhemoglobin absorbs wavelengths of the red spectrum and allows wavelengths in the infrared spectrum to transmit through the molecule. The spectrophotometer in the pulse oximeter transmits light in the red (660 nanometers) and infrared (920 nanometers) spectra. The different wavelengths of light are transmitted across a pulsatile vascular bed and are detected by a photodetector on the other side. The photodetector processes the amount of light of varying wavelengths that reaches it, then transmits an electrical current to a processor that calculates the difference in the amount of light originally transmitted and the amount of light of similar wavelength that actually reaches the photodetector. The difference in each reflects the amount of light absorbed in the pulsatile blood and can be used to calculate the amount or ratio of oxyhemoglobin to deoxyhemoglobin in circulation, or the functional hemoglobin saturation by the formula:

$$SaO_2 = HbO_2 / HbO_2 + Hb$$

where HbO_2 is oxygenated hemoglobin, and Hb is deoxygenated hemoglobin. Four molecules of oxygen reversibly bind to hemoglobin for transport to the tissues. Carbon monoxide similarly binds to hemoglobin and forms carboxyhemoglobin, a molecule that is detected similarly as oxygenated hemoglobin. Thus SaO_2 as detected by a pulse oximeter is not reliable if carboxyhemoglobin is present.

In most cases, pulse oximetry or SaO_2 corresponds reliably to the oxyhemoglobin dissociation curve. Oxygen saturation greater than 90% corresponds to a PaO_2 greater than 60 mm Hg. Above this value, large changes in PaO_2 are reflected in relatively small changes in SaO_2 , making pulse oximetry a relatively insensitive method of determining oxygenation status when PaO_2 is normal.

Because pulse oximetry measures oxygenated versus nonoxygenated hemoglobin in pulsatile blood flow, it is fairly unreliable when severe vasoconstriction, hypothermia, shivering or trembling, or excessive patient movement are present. Additionally, increased ambient lighting and the presence of methemoglobin or carboxyhemoglobin also can cause artifactual changes in the SaO_2 , and thus the measurement is not reliable or accurate. Most pulse oximeters also display a waveform and the patient's heart rate. If the photodetector does not detect a good quality signal, the waveform will not be normal, and the heart rate displayed on the monitor will not correlate with the patient's actual heart rate.

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CAPNOMETRY (END-TIDAL CARBON DIOXIDE MONITORING)

The efficiency of ventilation is evaluated using the PaCO_2 value on an arterial blood gas sample. Alternatively, a noninvasive method to determine end-tidal carbon dioxide is through use of a capnograph. The science of capnometry uses a spectrophotometer to measure carbon dioxide levels in exhaled gas. The capnometer is placed in the expiratory limb of an anesthetic circuit. A sample of exhaled gas is aliquoted from the breath, and an infrared light source is passed across the sample. A photodetector on the other side of the sample flow measures the amount or concentration of carbon dioxide in the sample of expired gas. The calculated value is displayed as end-tidal carbon dioxide. This value also can be displayed as a waveform.

When placed in graphic form, a waveform known as a capnograph is displayed throughout the ventilatory cycle. Normally, at the onset of exhalation, the gas exhaled into the expiratory limb of the tubing comes from the upper airway or physiologic dead space and contains relatively little carbon dioxide. As exhalation continues, a steep uphill slope occurs as more carbon dioxide is exhaled from the bronchial tree. Near the end of exhalation, the capnogram reaches a plateau, which most accurately reflects the carbon dioxide level at the level of the alveolus. Because carbon dioxide diffuses across the alveolar basement membrane so rapidly, this reflects arterial carbon dioxide levels. If a plateau is not reached and notching of the waveform occurs, check the system for leaks. If the baseline waveform does not reach zero, the patient may be rebreathing carbon dioxide or may be tachypneic, causing physiologic positive end-expiratory pressure. The soda-sorb in the system should be replaced if it has expired. Conversely, low end-tidal carbon dioxide may be associated with a decrease in perfusion or blood flow. Decreased perfusion can be associated with low end-tidal carbon dioxide values, particularly during cardiopulmonary cerebral resuscitation. End-tidal carbon dioxide levels are one of the most accurate predictors of the efficacy of cardiopulmonary cerebral resuscitation and patient outcome. Additionally, the difference between arterial carbon dioxide levels (PaCO_2) and end-tidal carbon dioxide can be used to calculate dead-space ventilation. Increases in the difference also occur with poor lung perfusion and pulmonary diffusion impairment.

Additional Reading

- Day TK: Blood gas analysis, *Vet Clin North Am Small Anim Pract* 32:1031-1048, 2002.
- Hackett TB: Pulse oximetry and end-tidal carbon dioxide monitoring, *Vet Clin North Am Small Anim Pract* 32:1021-1029, 2002.
- Hendricks JC, King LG: Practicality, usefulness, and limits of pulse oximetry in critical small animal patients, *J Vet Emerg Crit Care* 3:5-12, 1993.
- Marino PL: Oximetry and capnography. In *The ICU book*, ed 2, Baltimore, 1998, Williams & Wilkins.
- Proulx J: Respiratory monitoring: arterial blood gas analysis, pulse oximetry, and end-tidal carbon dioxide analysis, *Clin Tech Small Anim Pract* 14:227-230, 1999.
- Wright B, Hellyer PW: Respiratory monitoring during anesthesia: pulse oximetry and capnography, *Compend Contin Educ Pract Vet* 18:1083-1097, 1996.

THORACOCENTESIS

Thoracocentesis refers to the aspiration of fluid or air from within the pleural space. Thoracocentesis may be diagnostic to determine whether air or fluid is present and to characterize the nature of the fluid obtained. Thoracocentesis also can be therapeutic when removing large volumes of air or fluid to allow pulmonary reexpansion and correction of hypoxemia and orthopnea.

To perform thoracocentesis, follow this procedure:

1. First, assemble the equipment necessary (Box 1-16).
2. Next, clip a 10-cm square in the center of the patient's thorax on both sides.
3. Aseptically scrub the clipped area.
4. Ideally, thoracocentesis should be performed within the seventh to ninth intercostal space. Rather than count rib spaces in an emergent situation, visualize the thoracic

BOX 1-16 EQUIPMENT REQUIRED FOR THORACOCENTESIS

- 22- to 20-inch over-the-needle catheters or hypodermic needles
- 60-mL catheter-tipped syringe
- Intravenous extension tubing
- Three-way stopcock
- Clippers
- Antimicrobial scrub
- Latex gloves

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cage as a box, and the clipped area as a box within the box. You will insert your needle or catheter in the center of the box and then direct the bevel of the needle dorsally or ventrally to penetrate pockets of fluid or air present.

5. Attach the needle or catheter hub to the length of intravenous extension tubing. Attach the female port of the intravenous extension tubing to the male port of the three-way stopcock. Attach the male port of the 60-mL syringe to one of the female ports of the three-way stopcock. The apparatus is now assembled for use.
6. Insert the needle through the intercostal space such that the bevel of the needle initially is directed downward.
7. Next, push down on the hub of the needle such that the needle becomes parallel with the thoracic wall. By moving the hub of the needle in a clockwise or counterclockwise manner, the bevel of the needle will move within the thoracic cavity to penetrate pockets of air or fluid. In general, air is located dorsally and fluid is located more ventrally, although this does not always occur.
8. Aspirate air or fluid. Save any fluid obtained for cytologic and biochemical analyses and bacterial culture and susceptibility testing. In cases of pneumothorax, if the thoracocentesis needs to be repeated more than 3 times, consider using a thoracostomy tube.

THORACOSTOMY TUBE

Place a thoracostomy tube in cases of pneumothorax whenever negative suction cannot be obtained or repeated accumulation of air requires multiple thoracocentesis procedures. Thoracostomy tubes also can be placed to drain rapidly accumulating pleural effusion and for the medical management of pyothorax. Before attempting thoracostomy tube placement, make sure that all necessary supplies are assembled (Box 1-17; Table 1-13).

To place a thoracostomy tube, follow this procedure:

1. Lay the patient in lateral recumbency.
2. Clip the patient's entire lateral thorax.
3. Aseptically scrub the lateral thorax.
4. Palpate the tenth intercostal space.
5. Have an assistant pull the patient's skin cranially and ventrally toward the point of the elbow. This will facilitate creating a subcutaneous tunnel around the thoracostomy tube.
6. Draw up 2 mg/kg 2% lidocaine (1 mg/kg for cats) along with a small amount of sodium bicarbonate to take away some of the sting.
7. Insert the needle at the dorsal aspect of the tenth intercostal space and to the seventh intercostal space. Inject the lidocaine into the seventh intercostal space at the point where the trocarized thoracic drainage catheter will penetrate into the thoracic cavity. Slowly infuse the lidocaine as you withdraw the needle to create an anesthetized tunnel through which to insert the catheter.
8. While the local anesthetic is taking effect, remove the trocar from the catheter and cut the proximal end of the catheter with a Mayo scissors to facilitate adaptation with the Christmas tree adapter.

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BOX 1-17 SUPPLIES REQUIRED FOR PLACEMENT OF A THORACOSTOMY TUBE

- Argyle trocar thoracic drainage catheter
- Three-way stopcock
- 22-gauge orthopedic wire
- No. 10 scalpel blade
- Needle holder (sterile)
- Sterile huck towels
- Thumb forceps (sterile)
- 2-0 to 0 nonabsorbable suture
- Gauze 4 × 4-inch squares (sterile)
- Clippers
- Clear adhesive antimicrobial barrier drape
- Christmas tree adapter
- Intravenous extension tubing
- Mayo scissors (sterile)
- Scalpel handle (sterile)
- 2% lidocaine
- Towel clamps
- 25-gauge hypodermic needle
- 3- to 6-mL syringe
- Cotton roll gauze
- Elastikon
- Sterile gloves

TABLE 1 - 13 Size of Dog or Cat and Appropriate Chest Catheter Size

Dog/Cat size	Catheter
<7 kg	14-16 F
7-15 kg	18-22 F
16-30 kg	22-28 F
>30 kg	28-36 F

9. Attach the Christmas tree adapter to the three-way stopcock and the three-way stopcock to a length of intravenous extension tubing and the 60-mL syringe so that the apparatus can be attached immediately to the thoracostomy tube after placement.
10. Aseptically scrub the lateral thorax a second time and then drape it with sterile huck towels secured with towel clamps.
11. Wearing sterile gloves, make a small stab incision at the dorsal aspect of the tenth intercostal space.
12. Insert the trocar back into the thoracostomy drainage tube. Insert the trocar and tube into the incision. Tunnel the tube cranially for approximately 3 intercostal spaces while an assistant simultaneously pulls the skin cranially and ventrally toward the point of the elbow.
13. At the seventh intercostal space, direct the trocar and catheter perpendicular to the thorax. Grasp the catheter apparatus at the base adjacent to the thorax to prevent the trocar from going too far into the thorax.
14. Place the palm of your dominant hand over the end of the trocar, and push the trocar and catheter into the thoracic cavity, throwing your weight into the placement in a swift motion, not by banging the butt of your hand on the end of the stylette. For small individuals, standing on a stool, or kneeling over the patient on the triage table can create leverage and make this process easier. The tube will enter the thorax with a pop.
15. Gently push the catheter off of the stylette, and remove the stylette.
16. Immediately attach the Christmas tree adapter and have an assistant start to withdraw air or fluid while you secure the tube in place.
17. First, place a horizontal mattress suture around the tube to cinch the skin securely to the tube. Use care to not penetrate the tube with your needle and suture.
18. Next, place a purse-string suture around the tube at the tube entrance site. Leave the ends of the suture long, so that you can create a finger-trap suture to the tube, holding the tube in place.

19. Place a large square of antimicrobial-impregnated adhesive tape over the tube for further security and sterility.
20. If antimicrobial adhesive is not available, place a gauze pad 4 × 4 inches square over the tube, and then wrap the tube to the thorax with cotton roll gauze and Elastikon adhesive tape.
21. Draw the location of the tube on the bandage to prevent cutting it with subsequent bandage changes.

An alternate technique to use if a trocar thoracic drainage catheter is not available is the following:

1. Prepare the lateral thorax and infuse local lidocaine anesthetic as listed before.
2. Make a small stab incision with a No. 10 scalpel blade, as listed before.
3. Obtain the appropriately sized red rubber catheter and cut multiple side ports in the distal end of the catheter, taking care to not cut more than 50% of the circumference of the diameter of the tube.
4. Insert a rigid, long urinary catheter into the red rubber catheter to make the catheter more rigid during insertion into the pleural space.
5. Grasp the distal end of the catheter(s) in the teeth of a large Carmalt. Tunnel a Metzenbaum scissors under the skin to the seventh intercostal space and make a puncture through the intercostal space.
6. Remove the Metzenbaum scissors, and then tunnel the Carmalt and red rubber tube under the skin to the hole created in the seventh intercostal space with the Metzenbaum scissors.
7. Insert the tips of the Carmalt and the red rubber catheter through the hole, and then open the teeth of the Carmalt.
8. Push the red rubber catheter cranially into the pleural cavity.
9. Remove the Carmalt and the rigid urinary catheter, and immediately attach the suction apparatus. Secure the red rubber catheter in place as listed before.

Additional Reading

- Mazzaferro EM: Pulmonary injury secondary to trauma. In Wingfield WE, Raffae MR, editors: *The veterinary ICU book*, Jackson, Wyo, 2002, Teton NewMedia.
- Tseng LW, Waddell LS: Approach to the patient in respiratory distress, *Clin Tech Small Anim Pract* 15(2):53-62, 2000.

TRACHEOSTOMY

Placement of a temporary tracheostomy can be lifesaving to relieve upper respiratory tract obstruction, to facilitate removal of airway secretions, to decrease dead space ventilation, to provide a route of inhalant anesthesia during maxillofacial surgery, and to facilitate mechanical ventilation.

In an emergent situation in which asphyxiation is imminent and endotracheal intubation is not possible, any cutting instrument placed into the trachea distal to the point of obstruction can be used. To perform a slash tracheostomy, quickly clip the fur and scrub the skin over the third tracheal ring. Make a small cut in the trachea with a No. 11 scalpel blade, and insert a firm tube, such as a syringe casing. Alternately, insertion of a 22-gauge needle attached to intravenous extension tubing and adapted with a 1-mL syringe case to attach to a humidified oxygen source also temporarily can relieve obstruction until a temporary tracheostomy can be performed.

In less emergent situations, place the patient under general anesthesia and intubate the patient. Assemble all the equipment necessary before starting the temporary tracheostomy procedure (Box 1-18).

To perform a tracheostomy, follow this procedure:

1. Place the patient in dorsal recumbency.
2. Clip the ventral cervical region from the level of the ramus of the mandible caudally to the thoracic inlet and dorsally to midline.

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BOX 1-18 SUPPLIES REQUIRED FOR A TRACHEOSTOMY

- Sterile huck towels
- Towel clamps
- Antimicrobial scrub
- No. 10 scalpel blade
- Curved mosquito hemostats
- Metzenbaum scissors
- Thumb forceps
- 3-0 to 2-0 nonabsorbable suture material
- Needle holders
- Shiley low-pressure cuff tracheostomy tube OR endotracheal tube that has been cut and adapted to create a tracheostomy tube
- Umbilical tape

3. Aseptically scrub the clipped area, and then drape with sterile huck towels secured with towel clamps.
4. Make a 3-cm ventral midline skin incision over the third to sixth tracheal rings, perpendicular to the trachea.
5. Bluntly dissect through the sternohyoid muscles to the level of the trachea.
6. Carefully pick up the fascia overlying the trachea and cut it away with a Metzenbaum scissors.
7. Place two stay sutures through/around adjacent tracheal rings.
8. Incise in between trachea rings with a No. 11 scalpel blade. Take care to not cut more than 50% of the circumference of the trachea.
9. Using the stay sutures, pull the edges of the tracheal incision apart, and insert the tracheostomy tube. The Shiley tube contains an internal obturator to facilitate placement into the tracheal lumen. Remove the obturator, and then insert the inner cannula, which can be removed for cleaning as needed.
10. Once the tube is in place, secure the tube around the neck with a length of sterile umbilical tape.

TRACHEOSTOMY TUBE CARE

Postoperative care of the tracheostomy tube is as important as the procedure itself. Because the tracheostomy tube essentially bypasses the protective effects of the upper respiratory system, one of the most important aspects of tracheostomy tube care and maintenance is to maintain sterility at all times. Any oxygen source should be humidified with sterile water or saline to prevent drying of the respiratory mucosa. If supplemental oxygen is not required, instill 2 to 3 mL of sterile saline every 1 to 2 hours to moisten the mucosa. Wearing sterile gloves, remove the internal tube and place it in a sterile bowl filled with sterile hydrogen peroxide and to be cleaned every 4 hours (or more frequently as necessary). If a Shiley tube is not available, apply suction to the internal lumen of the tracheostomy tube every 1 to 2 hours (or more frequently as needed) with a sterile 12F red rubber catheter attached to a vacuum pump to remove any mucus or other debris that potentially could plug the tube. Unless the patient demonstrates clinical signs of fever or infection, the prophylactic use of antibiotics is discouraged because of the risk of causing a resistant infection. After the temporary tracheostomy is no longer necessary, remove the tube and sutures, and leave the wound to heal by second intention. Primary closure of the wounds could predispose the patient to subcutaneous emphysema and infection.

Additional Reading

- Baker GD: Trans-tracheal oxygen therapy in dogs with severe respiratory compromise due to tick (*I. holocyclus*) toxicity, *Aust Vet Pract* 34(2):83, 2004.
- Colley P, Huber M, Henderson R: Tracheostomy techniques and management, *Compend Contin Educ Pract Vet* 21(1):44-53, 1999.
- Hedlund CS: Surgery of the upper respiratory system. In Fossum TW, editor: *Small animal surgery*, St Louis, 2002, Mosby.
- Hedlund CS: Tracheostomies in the management of canine and feline upper respiratory disease, *Vet Clin North Am Small Anim Pract* 24(5):873-886, 1994.

UROHYDROPULSION

Urohydropulsion is a therapeutic procedure for removal of uroliths from the urethra of the male dog. The technique works best if the animal is heavily sedated or is placed under general anesthesia (Figure 1-12).

To perform urohydropulsion, follow this procedure:

1. Place the animal in lateral recumbency.
2. Clip the fur from the distal portion of the prepuce.
3. Aseptically scrub the prepuce and flush the prepuce with 12 to 20 mL of antimicrobial flush solution.
4. Have an assistant who is wearing gloves retract the penis from the prepuce.
5. While wearing sterile gloves, lubricate the tip of a rigid urinary catheter as for urethral catheterization.
6. Gently insert the tip of the catheter into the urethra until you meet the resistance of the obstruction.
7. Pinch the tip of the penis around the catheter.
8. Have an assistant insert a gloved lubricated finger into the patient's rectum and press ventrally on the floor of the rectum to obstruct the pelvic urethra.
9. Attach a 60-mL syringe filled with sterile saline into proximal tip of the catheter.
10. Quickly inject fluid into the catheter and alternate compression and relaxation on the pelvic urethra such that the urethra dilates and suddenly releases the pressure, causing dislodgement of the stone. Small stones may be ejected from the tip of the urethra, whereas larger stones may be retropulsed back into the urinary bladder to be removed surgically at a later time.

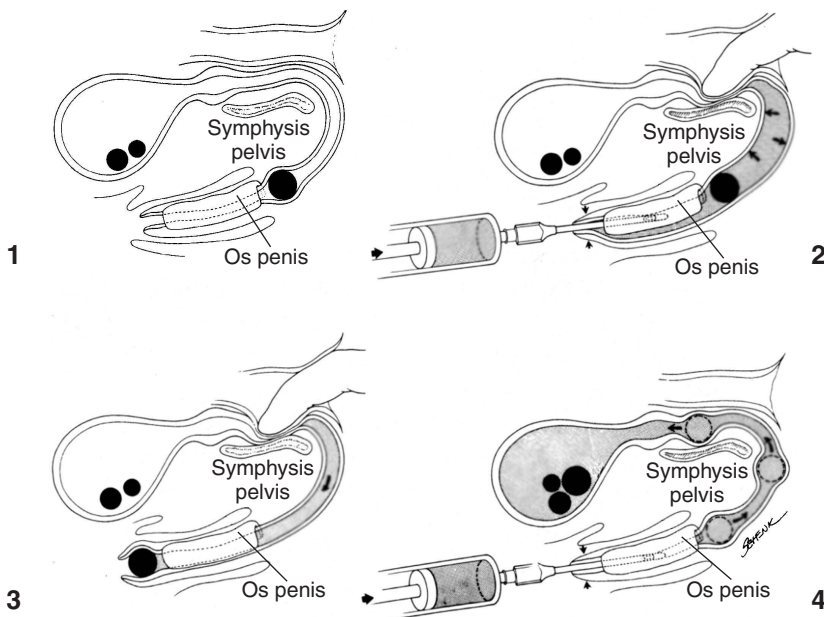


Figure 1-12: Removal of urethrolith in a male dog by urohydropulsion: 1, Urethrolith originating from the urinary bladder has lodged behind the os penis. 2, Dilation of the urethral lumen is achieved by injecting fluid with pressure. Digital pressure applied to the external urethral orifice and the pelvic urethra has created a closed system. 3, Sudden release of digital pressure at the external urethral orifice and subsequent movement of fluid and urethrolith toward the urinary bladder. 4, Sudden release of digital pressure at the pelvic urethra and subsequent movement of fluid and urethrolith toward the external urethral orifice.

(From Osborne CA, Finco DR: Canine and Feline Nephrology and Urology. Williams and Wilkins, Baltimore, 1995.)

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Additional Reading

Osborne CA, Finco DR: *Canine and feline nephrology and urology*, Baltimore, 1995, Williams & Wilkins.

VASCULAR ACCESS TECHNIQUES

The type of catheter that you choose for vascular access depends largely on the size and species of the patient, the fragility of the vessels to be catheterized, the proposed length of time that the catheter will be in place, the type and viscosity of the fluid or drug to be administered, the rate of fluid flow desired, and whether multiple repeated blood samples will be required (Table 1-14).

A variety of over-the-needle, through-the-needle, and over-the-wire catheters are available for placement in a variety of vessels, including the jugular, cephalic, accessory cephalic, medial saphenous, lateral saphenous, dorsal pedal artery, and femoral artery.

One of the most important aspects of proper catheter placement and maintenance is to maintain cleanliness at all times. The patient’s urine, feces, saliva, and vomit are common sources of contamination of the catheter site. Before placing a peripheral or central catheter in any patient, consider the patient’s physical status including whether vomiting, diarrhea, excessive urination, or seizures. In a patient with an oral mass that is drooling excessively or a patient that is vomiting, peripheral cephalic catheterization may not be the most appropriate, to prevent contamination. Conversely, in a patient with excessive urination or diarrhea, a lateral or medial saphenous catheter is likely to become contaminated quickly.

Whenever one places or handles a catheter or intravenous infusion line, the person should wash the hands carefully and wear gloves to prevent contamination of the intravenous catheter and fluid lines. One of the most common sources of catheter contamination in veterinary hospitals is through caretakers’ hands. In emergent situations, placement of a catheter may be necessary under less than ideal circumstances. Remove those catheters as soon as the patient is more stable, and place a second catheter using aseptic techniques.

In general, once the location of the catheter has been decided, set up all equipment necessary for catheter placement before starting to handle and restrain the patient. Box 1-19 lists the equipment needed for most types of catheter placement.

TABLE 1-14 Catheter Sizes for Vascular Access		
	Cephalic or tarsal vein (catheter gauge)	Jugular (catheter gauge)
Cat or small dog	20-24	16-18
Medium-sized dog	18-22	16-18
Large dog	14-20	14-18

BOX 1-19 EQUIPMENT NECESSARY FOR INTRAVENOUS CATHETER PLACEMENT

- Antimicrobial ointment
- Antimicrobial scrub
- Cotton ball
- Electric clippers and No. 40 blade
- Gauze, 4 × 4-inch squares
- Heparinized saline flush
- Intravenous catheter
- 1/2- and 1-inch white adhesive tape
- Male adapter or T port flushed with heparinized saline

After setting up all of the supplies needed, clip the fur over the site of catheter placement. Make sure to clip all excess fur and long feathers away from the catheter site, to prevent contamination. For catheter placement in limbs, clip the fur circumferentially around the site of catheter placement to facilitate adherence of the tape to the limb and to facilitate catheter removal with minimal discomfort at a later date. Next, aseptically scrub the catheter site with an antimicrobial scrub solution such as Hibiclens. The site is now ready for catheter insertion.

CENTRAL VENOUS CATHETERS

Consider using a central venous catheter whenever multiple repeated blood samples will need to be collected from a patient during the hospital stay. Central venous catheters also can be used for CVP measurement, administration of hyperoncotic solutions such as parenteral nutrition, and administration of crystalloid and colloid fluids, anesthesia, and other injectable drugs (Figures 1-13 and 1-14).

Percutaneous through-the-needle jugular catheter placement

To place a jugular central venous catheter, place the patient in lateral recumbancy and extend the head and neck such that the jugular furrow is straight. Clip the fur from the ramus of the mandible caudally to the thoracic inlet and dorsally and ventrally to midline. Wipe the clipped area with gauze 4 × 4-inch squares to remove any loose fur and other debris. Aseptically scrub the clipped area with an antimicrobial cleanser.

Venocaths (Abbott Laboratories) are a through-the-needle catheter that is contained within a sterile sleeve for placement. Alternately, other over-the-wire central venous catheters can be placed by the Seldinger technique. Sterility must be maintained at all times, regardless of the type of catheter placed.

Wearing sterile gloves, drape the site of catheter placement with sterile drapes, and occlude the jugular vein at the level of the thoracic inlet. Pull the clear ring and wings of



Figure 1-13: Lateral thoracic radiograph of a central venous catheter. Note that the tip of the catheter is inserted in its proper location, just outside of the right atrium.

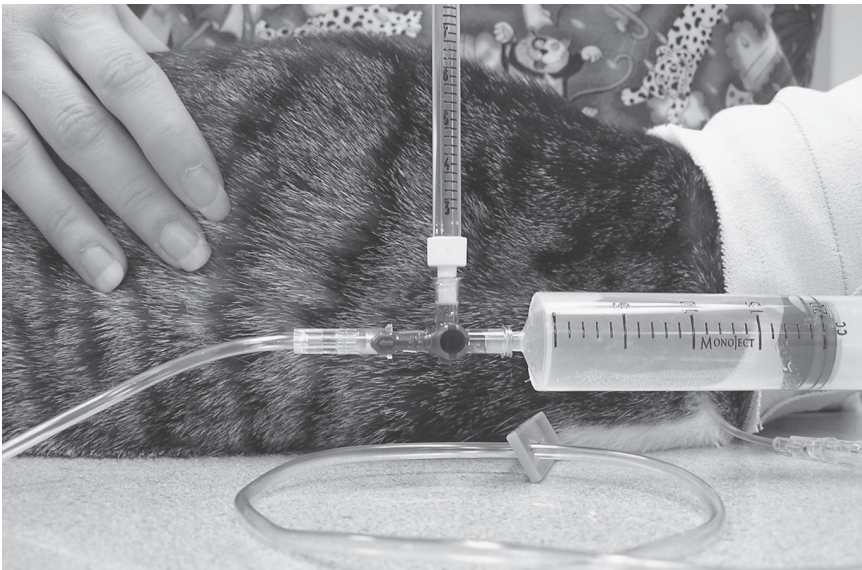


Figure 1-14: Measuring the patient's central venous pressure (CVP). Note that the 0 marker on the manometer is at the patient's manubrium.

the catheter cover down toward the catheter itself to expose the needle. Remove the guard off of the needle. Lift the skin over the proposed site of catheter insertion and insert the needle under the skin, with the bevel of the needle facing up. Next, reocclude the vessel and pull the skin tight over the vessel to prevent movement of the vessel as you attempt to insert the needle. In some cases, it may be difficult actually to see the vessel in obese patients. If you cannot visualize or palpate the needle, gently bounce the needle over the vessel with the bevel up. The vessel will bounce in place slightly, allowing a brief moment of visualization to facilitate catheter placement. Once the vessel has been isolated and visualized, insert the needle into the vessel at a 15- to 30-degree angle. Watch closely for a flash of blood in the catheter. When blood is observed, insert the needle a small distance farther, and then push the catheter and stylette into the vessel for the entire length, until the catheter and stylette can be secured in the catheter hub. If the catheter cannot be inserted fully into the vessel for its entire length, the tip of the needle may not be within the entire lumen, the catheter may be directed perivascularly, and the catheter may be caught at the thoracic flexure and may be moving into one of the tributaries that feeds the forelimb. Extend the patient's head and neck, and lift the forelimb up to help facilitate placement. Do not force the catheter in because the catheter potentially can form a knot and will need to be removed surgically. Remove the needle from the vessel, and have an assistant place several 4 × 4-inch gauze squares over the site of catheter placement with some pressure to control hemorrhage. Secure the catheter hub into the needle guard, and remove the stylette from the catheter. Immediately insert a 3- to 6-mL syringe of heparinized saline and flush the catheter and draw back. If you are in the correct place, you will be able to draw blood from the catheter.

To secure the catheter in place, tear a length of 1-inch white tape that will wrap around the patient's neck. Pull a small length of the catheter out of the jugular vein to make a semicircle. The semicircle should be approximately ½ inch in diameter. Let the length of catheter lie on the skin, and then place 4 × 4-inch gauze squares impregnated with antimicrobial ointment over the site of catheter insertion. Secure the proximal end of white tape around the white and blue pieces of the catheter, and wrap the tape around the patient's neck so that the tape adheres to the skin and fur. Repeat the process by securing the gauze to the skin with two additional lengths of white tape, starting to secure the gauze in place

by first wrapping the tape dorsally over the patient's neck, rather than under the patient's neck. In between each piece of tape and bandage layer, make sure that the catheter flushes and draws back freely, or else occlusion can occur. Gently wrap layers of cotton roll gauze, Kling, and Elastikon or Vetrap over the catheter. Secure a male adapter or T port that has been flushed with heparinized saline, and then label the catheter with the size and length of catheter, date of catheter placement, and initials of the person who placed the catheter. The catheter is ready for use. Monitor the catheter site daily for erythema, drainage, vessel thickening, or pain upon infusion. If any of these signs occur, or if the patient develops a fever of unknown origin, remove the catheter, culture the catheter tip aseptically, and replace the catheter in a different location. As long as the catheter is functional without complications, the catheter can remain in place.

Percutaneous over-the-wire jugular catheter placement (Seldinger technique)

Central catheters also can be placed via the Seldinger or over-the-wire technique. A number of companies manufacture kits that contain the supplies necessary for over-the-wire catheter placement. Each kit minimally should contain an over-the-needle catheter to place into the vessel, a long wire to insert through the original catheter placed, a vascular dilator to dilate the hole in the vessel created by the first catheter, and a long catheter to place into the vessel over the wire. Additional accessories can include a paper drape, sterile gauze, a scalpel blade, local anesthetic, 22-gauge needles, and 3- or 6-mL syringes.

Restrain the patient and prepare the jugular furrow aseptically as for the percutaneous through-the-needle catheter placement. The person placing the catheter should wear sterile gloves throughout the process to maintain sterility. Pick up the skin over the site of catheter placement, and insert a small bleb of local anesthetic through the skin. The local anesthetic should not be injected into the underlying vessel (Figure 1-15). Make a small



Figure 1-15: Infusion of local anesthetic. Before making a nick incision through the skin, insert a bleb of lidocaine over the proposed site of catheter insertion. Pick up the skin to avoid intravenous injection of the anesthetic.

1

nick into the skin through the local anesthetic with a No. 10 or No. 11 scalpel blade. Use care to avoid lacerating the underlying vessel. Next, occlude the jugular vein as previously described, and insert the over-the-needle catheter into the vessel. Watch for a flash of blood in the catheter hub. Remove the stylette from the catheter. Next, insert the long wire into the catheter and into the vessel (Figures 1-16 and 1-17). Never let go of the wire. Remove the catheter, and place the vascular dilator over the wire and into the vessel (Figure 1-18). Gently twist to place the dilator into the vessel a short distance, creating a larger hole in the vessel. The vessel will bleed more after creating a larger hole. Remove the vascular dilator, and leave the wire in place within the vessel. Insert the long catheter over the wire into the vessel (Figure 1-19). Push the catheter into the vessel to the catheter hub (Figure 1-20). Slowly thread the wire through a proximal port in the catheter. Once the catheter is in place, remove the wire, and suture the catheter in place to the skin with nonabsorbable suture. Cover the catheter site with sterile gauze and antimicrobial ointment, cotton roll bandaging material, gauze, and Kling or Vetrap. Flush the catheter with heparinized saline solution, and then use the catheter for infusion of parenteral nutrition, blood products, crystalloid and colloid fluids, medications, and frequent blood sample collection. Examine the catheter site daily for evidence of infection or thrombophlebitis. The catheter can remain in place as long as it functions and no complications occur.

PERIPHERAL ARTERIAL AND VENOUS CATHETER PLACEMENT**Cephalic catheterization**

Place the patient in sternal recumbency as for cephalic venipuncture. Clip the antebrachium circumferentially, and wipe the area clean of any loose fur and debris (Figure 1-21). Aseptically scrub the clipped area, and have an assistant occlude the cephalic vein at the crook of the elbow. The person placing the catheter should grasp the distal carpus with the nondominant hand and insert the over-the-needle catheter into the vessel at a 15- to 30-degree angle (Figure 1-22). Watch for a flash of blood in the catheter hub, and then gently push the

Text continued on p. 66.

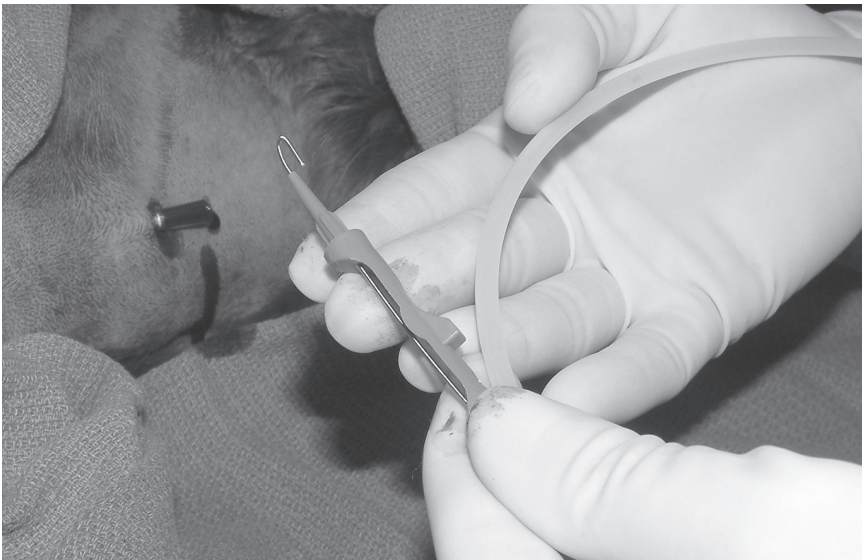


Figure 1-16: The J-Wire. The J-wire is curved at its tip to prevent iatrogenic trauma to the vessel and the heart. Pull the J-wire back so that the curve straightens out, and then insert the J-wire into the vessel.



Figure 1-17: Feeding the J-wire through the catheter. Insert the J-wire through the over-the-needle catheter into the vessel and then remove the over-the-needle catheter, leaving the J-wire in place. Never let go of the J-wire!



Figure 1-18: Insert the vascular dilator over the wire into the vessel with a twisting motion, to enlarge the hole in the vessel for ease of catheter placement later.

1



Figure 1-19: Feed the multi-lumen catheter over the wire that has already been inserted into the patient's vessel. Remember to never let go of the wire. The wire will eventually emerge from an open port in the proximal portion of the catheter, allowing its removal after the catheter has been seated in the vessel.

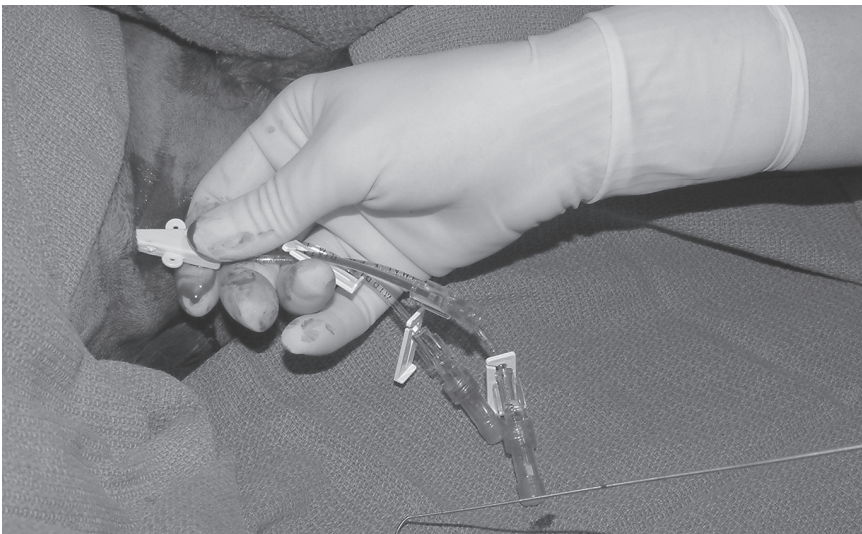


Figure 1-20: Catheter in vessel. The catheter is now seated in the patient's jugular vein, where it can be secured to the skin with nonabsorbable suture and then bandaged.



Figure 1-21: Clip the patient's antebra circumferentially to allow proper placement of the cephalic intravenous catheter.

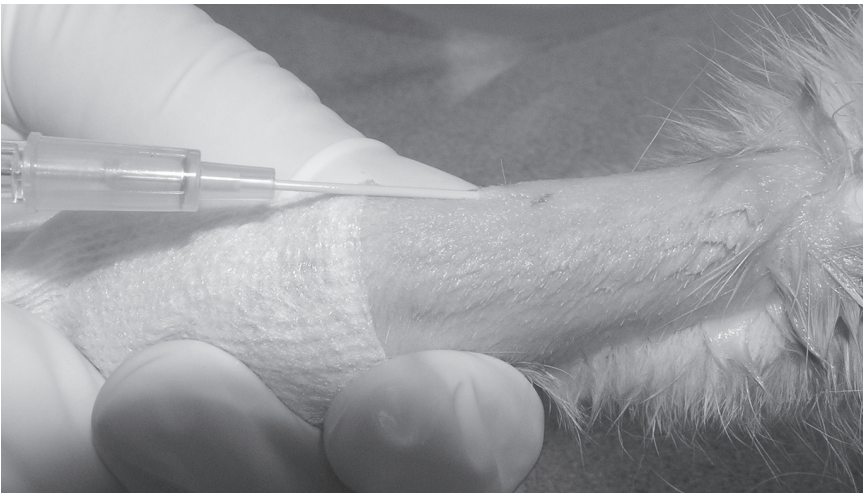


Figure 1-22: Insert the catheter through the skin into the vessel, watching for a flash of blood in the catheter hub.

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catheter off of the stylette (Figure 1-23). Have the assistant occlude the vessel over the catheter to prevent backflow. Flush the catheter with heparinized saline solution. Make sure that the skin and catheter hub are clean and dry to ensure that the tape adheres to the catheter hub and skin. Secure a length of 1/2-inch white tape tightly around the catheter and then around the limb. Make sure that the catheter hub does not “spin” in the tape, or else the catheter will fall out. Next, secure a second length of 1-inch adhesive tape under the catheter and around the limb and catheter hub (Figure 1-24). This piece of tape helps to stabilize the catheter in place. Finally, place a flushed T port or male adapter in the catheter hub and secure to the limb with white tape. Make sure that the tape is adhered to the skin



Figure 1-23: Blood in the catheter hub.



Figure 1-24: Secure the catheter hub to the patient's skin with a strip of 1/2-inch white tape.

securely, but not so tightly as to impede venous outflow (Figure 1-25). The catheter site can be covered with a cotton ball impregnated with antimicrobial ointment and layers of bandage material. Label all catheters with the date of placement, the type and gauge of catheter inserted, and the initials of the person who placed the catheter.

Percutaneous femoral artery catheterization

The femoral artery can be catheterized for placement of an indwelling arterial catheter. Indwelling arterial catheters can be used for continuous invasive arterial blood pressure monitoring and for procurement of arterial blood samples. Place the patient in lateral recumbancy, and tape the down leg in an extended position. Clip the fur over the femoral artery and aseptically scrub the clipped area. Palpate the femoral artery as it courses distally on the medial surface of the femur and anterior to the pectineus muscle. Make a small nick incision over the proposed site of catheter placement using the bevel of an 18-gauge needle. Place a long over-the-needle catheter through the nick in the skin and direct it toward the palpable pulse. Place the tip of the catheter so that the needle tip rests in the subcutaneous tissue between the artery and the palpating index finger. Advance the needle steeply at a 30-degree angle to secure the superficial wall of the vessel and then the deep wall of the vessel. The spontaneous flow of blood in the catheter hub ensures that the catheter is



Figure 1-25: Catheter is taped in place with a t-port.

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situated in the lumen of the artery. Feed the catheter off of the stylette, and cover the hub with a catheter cap. Flush the catheter with sterile heparinized saline solution, and then secure it in place. Some persons simply tape the catheter in place with pieces of ½- and 1-inch adhesive tape. Others use a “butterfly” piece of tape around the catheter hub and suture or glue the tape to the adjacent skin for added security.

Percutaneous dorsal pedal artery catheterization

The dorsal pedal artery commonly is used for catheter placement. To place a dorsal pedal arterial catheter, place the patient in lateral recumbency. Clip the fur over the dorsal pedal artery, and then aseptically scrub the clipped area. Tape the distal limb so that the leg is twisted slightly medially for better exposure of the vessel, or the person placing the arterial catheter can manipulate the limb into the appropriate position. Palpate the dorsal pedal pulse as it courses dorsally over the tarsus. Place an over-the-needle catheter percutaneously at a 15- to 30-degree angle, threading the tip of the needle carefully toward the pulse. Advance the needle in short, blunt movements, and watch the catheter hub closely for a flash of pulsating blood that signifies penetration into the lumen of the artery. Then thread the catheter off of the stylette, and cover the catheter hub with a catheter cap. Secure the catheter in place with lengths of ½- and 1-inch adhesive tape as with any other intravenous catheter, and then flush it with heparinized saline solution every 2 to 4 hours.

Surgical cutdown for arterial and venous catheter placement

Any vessel that can be catheterized percutaneously also can be catheterized with surgical cutdown. Restrain the patient and clip and aseptically scrub the limb or jugular vein as for a percutaneous catheterization procedure. Block the area for catheter placement with a local anesthetic before cutting the skin over the vessel with a No. 11 scalpel blade. While wearing sterile gloves, pick up the skin and incise the skin over the vessel. Direct the sharp edge of the blade upward to avoid lacerating the underlying vessel. Using blunt dissection, push the underlying subcutaneous fat and perivascular fascia away from the vessel with a mosquito hemostat. Make sure that all tissue is removed from the vessel. Using the mosquito hemostat, place two stay sutures of absorbable suture under the vessel. Elevate the vessel until it is parallel with the incision, and gently insert the catheter and stylette into the vessel. Secure the stay sutures loosely around the catheter. Suture the skin over the catheter site with nonabsorbable suture, and then tape and bandage the catheter in place as for percutaneous placement. Remove catheters placed surgically as soon as possible and exchange them for a percutaneously placed catheter to avoid infection and thrombophlebitis.

MAINTENANCE OF INDWELLING ARTERIAL AND VENOUS CATHETERS

The most important aspect of catheter maintenance is to maintain cleanliness and sterility at all times. An indwelling catheter can remain in place for as long as it is functional and no complications occur. Change the bandage whenever it becomes wet or soiled to prevent wicking of bacteria and debris from the environment into the vessel. Check the bandages and catheter sites at least once a day for signs of thrombophlebitis: erythema, vessel hardening or ropiness, pain upon injection or infusion, and discharge. Also closely examine the tissue around and proximal and distal to the catheter. Swelling of the paw can signify that the catheter tape and bandage are too tight and are occluding venous outflow. Swelling above the catheter site is characteristic of perivascular leakage of fluid and may signify that the catheter is no longer within the lumen of the vessel.

Remove the catheter if it is no longer functional, if there is pain or resistance upon infusion, if there is unexplained fever or leukocytosis, or if there is evidence of cellulitis, thrombophlebitis, or catheter-related bacteremia or septicemia. Aseptically culture the tip of the indwelling catheter for bacteria. Animals should wear Elizabethan collars or other forms of restraint if they lick or chew at the catheter or bandage.

Catheter patency may be maintained with constant fluid infusion or by intermittent flushing with heparinized saline (1000 units of unfractionated heparin per 250 to 500 mL

of saline) every 6 hours. Flush arterial catheters more frequently (every 2 hours). Disconnect intravenous connections only when absolutely necessary. Wear gloves whenever handling the catheter or connections. Label all fluid lines and elevate them off of the floor to prevent contamination. Date each fluid line and replace it once every 24 to 36 hours.

INTRAOSSEOUS CATHETER PLACEMENT

If an intravenous catheter cannot be placed because of small patient size, hypovolemia, hypothermia, or severe hypotension, needles can be placed into the marrow cavity of the femur, humerus, and tibia for intraosseous infusion of fluids, drugs, and blood products. This technique is particularly useful in small kittens and puppies and in exotic species. Contraindications to intraosseous infusion is in avian species (which have air in their bones), fractures, and sepsis, because osteomyelitis can develop. An intraosseous catheter is relatively easy to place and maintain but can cause patient discomfort and so should be changed to an intravenous catheter as soon as vascular access becomes possible.

To place an intraosseous catheter, clip and aseptically scrub the fur over the proposed site of catheter placement. The easiest place for intraosseous placement is in the intertrochanteric fossa of the femur. Inject a small amount of a local anesthetic through the skin and into the periosteum where the trocar or needle will be inserted. Place the patient in lateral recumbency, and grasp the leg in between your fingers, with the stifle braced against the palm of your hand. Push the stifle toward the abdomen (medially) to abduct the proximal femur away from the body. This will shift the sciatic nerve out of the way of catheter placement. Insert the tip of the needle through the skin and into the intertrochanteric fossa. Gently push with a simultaneous twisting motion, pushing the needle parallel with the shaft of the femur, toward your palm. You may feel a pop or decreased resistance as the needle enters the marrow cavity. Gently flush the needle with heparinized saline. If the needle is plugged with bone debris, remove the needle and replace it with a fresh needle of the same type and size in the hole that you have created. A spinal needle with an internal stylette also can be placed. The stylette will prevent the needle from becoming clogged with bone debris during insertion. Secure the hub of the needle with a butterfly length of white adhesive tape and then suture it to the skin to keep the catheter in place. The catheter is now ready for use. The patient should wear an Elizabethan collar to prevent disruption or removal of the catheter. The intraosseous catheter can be maintained as any peripheral catheter, with frequent flushing and daily evaluation of the catheter site.

Additional Reading

Beal MW, Hughes D: Vascular access: theory and techniques in the small animal emergency patient, *Clin Tech Small Anim Pract* 15(2):101-109, 2000.

Hansen BD: Technical aspects of fluid therapy. In DiBartola S, editor: *Fluid therapy in small animal practice*, Philadelphia, 2000, WB Saunders.

Otto CM, Kaufman GM, Crowe DT: Intraosseous infusion of fluids and therapeutics, *Compend Contin Educ Pract Vet* 11:421-430, 1989.

Shaw S, Walshaw S: *Manual of clinical procedures in the dog, cat, and rabbit*, ed 2, Philadelphia, 1997, Lippincott-Raven.

PAIN: ASSESSMENT, PREVENTION, AND MANAGEMENT*

The definition of pain has been debated philosophically over the ages and has changed as knowledge has increased. Pain is defined as an unpleasant sensory or emotional experience associated with actual or perceived tissue damage. Until recognition of a noxious stimulus occurs in the cerebral cortex, no response or adaptation results. Rational management of pain requires an understanding of the underlying mechanisms involved in pain and an appreciation of how analgesic agents interact to disrupt pain mechanisms.

*Contributed by A. Looney, B. Hansen, and E. Hardie.

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BOX 1-20 CATEGORIES AND CAUSES OF PAIN IN DOGS AND CATS

ACUTE PAIN

Trauma
Thermal burn
Postoperative
Musculoskeletal
Visceral/pleural

CHRONIC PAIN

Arthritis
Cancer
Neurologic: diabetes mellitus
Musculoskeletal
Sympathetic dystrophies

Multiple factors and causes produce pain in human beings and domestic animal species. The causes of pain, psychological and physical, may derive from many different mechanisms within emergency medicine, among them trauma, infectious disease, neglect, environmental stress, surgery, and acute decompensation of chronic medical conditions. The two major classes of pain are acute and chronic pain. Box 1-20 gives specific categories and causes of pain.

The pain sensing and response system can be divided into the following categories: *nociceptors*, which detect and filter the intensity of the noxious stimuli; *primary afferent nerves*, which transmit impulses to the central nervous system (CNS); *ascending tracts*, which are part of the dorsal horn and the spinal cord that conveys stimuli to higher centers in the brain; *higher centers*, which are involved in pain discrimination, some memory, and motor control; and *modulating or descending systems*, which are a means of processing, memorizing, and modifying incoming impulses. Current analgesic therapies may inhibit afferent nociceptive transmission within the brain and spinal cord; directly interrupt neural impulse conduction through the dorsal horn, primary afferent nerves, or dorsal root ganglion; or prevent the nociceptor sensitization that accompanies initial pain and inflammation. The physiologic aspects of pain are believed to be produced by the transmission, transduction, and integration of initial nerve endings, peripheral neuronal input, and ascending afferent nerves via the thalamus to the cerebral cortex. Ascending afferent nerves to the limbic system are believed to be responsible for the emotional aspects of pain.

There are several classification schemes for different types of pain. *Acute pain*, such as that which results from trauma, surgery, or infectious agents, is abrupt in onset, relatively short in duration, and may be alleviated easily by analgesics. In contrast, *chronic pain* is a long-standing physical disorder or emotional distress that is slow in onset and difficult to treat. Both types of pain can be classified further based on site of origin. *Somatic pain* arises from superficial skin, subcutaneous tissue, body wall, or appendages. *Visceral pain* arises from abdominal or thoracic viscera and primarily is associated with serosal irritation. *Analgesia*, then, is the loss of pain WITHOUT the loss of consciousness. This is in contrast to *anesthesia*, which is the loss of sensation in the whole body or a part of the body WITH the loss of consciousness or at least depression of the CNS.

PHYSIOLOGIC IMPACT OF UNTREATED PAIN

Untreated pain causes immediate changes in the neurohormonal axis, which in turn causes restlessness, agitation, increased heart and respiratory rates, fever, and blood pressure fluctuations, all of which are detrimental to the healing of the animal. A catabolic state is created as a result of increased secretion of catabolic hormones and decreased secretion of anabolic hormones. The net effect the majority of neurohormonal changes produce is an increase in the secretion of catabolic hormones. Hyperglycemia is produced and may persist because of production of glucagon and relative lack of insulin. Lipolytic activity is stimulated by cortisol, catecholamines, and growth hormone. Cardiorespiratory effects of pain include increased cardiac output, vasoconstriction, hypoxemia, and hyperventilation. Protein catabolism is a common occurrence and major concern regarding healing. Pain associated with inflammation causes increase in tissue and blood levels of prostaglandins

and cytokines, both of which promote protein catabolism indirectly by increasing the energy expenditure of the body.

Powerful evidence indicates that local anesthetic, sympathetic agonist, and opioid neural blockade may produce a modification of the responses to these physiologic changes. Variable reduction in plasma cortisol, growth hormone, antidiuretic hormone, β -endorphin, aldosterone, epinephrine, norepinephrine, and renin is based on the anesthetic technique and the drugs selected. Prophylactic administration of analgesics blunts the response before it occurs; analgesics administered following perception or pain are not as effective, and higher doses are generally necessary to achieve an equivalent level of analgesia.

RECOGNITION AND ASSESSMENT OF PAIN

Effective pain control can be achieved only when the signs of pain can be assessed effectively, reliably, and regularly. The experience of pain is unique to each individual, which makes pain assessment difficult, especially in traumatized and critical patients. Most attempts to assess clinical pain use behavioral observations and interactive variables in addition to assessment of physiologic responses such as heart rate and respiratory rate, blood pressure, and temperature. But many factors can influence the processing and outward projection of pain, including altered environments, species differences, within-species variations (age, breed, sex), and the type, severity, and chronicity of pain.

Within-species differences (age, breed, and sex) further complicate the pain assessment. Most notable is that different breeds of dogs act differently when confronted with pain or fear. Labrador Retrievers tend to be stoic, whereas Greyhounds and teacup breeds tend to react with a heightened state of arousal around even the simplest of procedures (e.g., subcutaneous injections and nail trims). The individual character and temperament of the animal further influences its response. Pediatric and neonatal animals seem to have a lower threshold for pain and anxiety than older animals. In any species, the duration and type of pain make it more (acute) or less (chronic) likely to be expressed or exhibited outwardly. Unfamiliarity with normal behaviors typical of a particular species or breed makes recognition of their painful behaviors and responses impossible.

The definition and recognition of pain in an individual animal is challenging. Because of all the differences discussed, there is no straight line from *insult*, albeit actual or perceived, to *degree* of pain experienced. Nor is there a formula for treating “X” type of pain with “Y” type of analgesic. A goal of analgesia is to treat all animals with analgesic drugs and modalities as PREEMPTIVELY as possible and using a multimodal approach. Use analgesic treatment as a tool for diagnosis of pain in the event that recognition of these phenomena is difficult for the patient. In other words, with countless drugs and treatment modalities available, analgesic administration should NEVER be withheld in an animal, even if pain is questionable.

PAIN ASSESSMENT IN DOGS AND CATS

It is important to remember that no behavior or physiologic variable in and of itself is pathognomonic for pain. Interactive and unprovoked (noninteractive) behavior assessments and trending of physiologic data are useful to determine the pain in an individual animal. This is known as *pain scoring*. Baseline observations, especially those observations from someone who has known the animal well, can be helpful to serial behavior and pain assessments. Pain scoring systems have been developed and are reviewed elsewhere; the purposes of these systems are to evaluate and to help guide diagnostic and analgesic treatments (Table 1-15). Regardless of the scale or method used to assess pain, the caregiver must recognize the limitations of the scale. If in doubt of whether pain is present or not, analgesic therapy should be used as a diagnostic tool.

BEHAVIORAL SIGNS OF ACUTE PAIN

Classic behaviors associated with pain in dogs and cats include abnormal postures, gaits, movements, and behaviors (Boxes 1-21 and 1-22). Stoicism is the apparent apathy and

TABLE 1 - 15 Pain Scale	
Number	Description
1	No pain
2	Mild pain
3	Moderate pain
4	Severe pain
5	Excruciating pain

BOX 1-21 BEHAVIORAL SIGNS ASSOCIATED WITH PAIN IN DOGS AND CATS	
ABNORMAL POSTURES Hunched-up Prayer position Inability to lie down Muscle atrophy (chronic) Reluctance to move Low tail carriage	ABNORMAL BEHAVIORS Focusing on area of pain (licking or chewing) Inappetance Lack of grooming Abnormal urination or defecation Stoicism Aggression Yawning Hiding Vocalizations Whimper Screaming/crying
ABNORMAL GAITS Stiffness Non-weight bearing Limping Pacing versus trotting Abnormal nail wear	
ABNORMAL MOVEMENTS Thrashing Restlessness Circling	

BOX 1-22 PHYSIOLOGIC SIGNS OF PAIN	
<ul style="list-style-type: none">• Acute pain• Allodynia• Blepharospasm• Bradycardia• Bruxism• Cardiac dysrhythmias• Hyperesthesia	<ul style="list-style-type: none">• Incontinence• Mydriasis• Panting• Ptyalism• Tachycardia• Tachypnea

indifference in the presence of pain and is perhaps the No. 1 sign of ineffective pain relief or persistent pain in many animals, because so many display apathy and classically normal physiologic parameters even in the face of severe distress, overt suffering, or blatant trauma and illness. The *absence of normal behaviors* is also a clinical sign of pain, even when abnormal behaviors are not observed.

SIGNS OF CHRONIC PAIN IN CATS AND DOGS

Acute pain results in many of the aforementioned behavioral and physiologic signs, but chronic pain in small animals is an entirely different and distinct entity. Chronic pain is often present in the absence of obvious tissue pathology and changes in physical demeanor.

Again, the severity of the pain may not correlate with the severity of any pathologic condition that may or may not be present. Chronic pain, especially if insidious in onset (cancer, dental, or degenerative pain), may well go unnoticed in dogs and cats, even by family members or intermittent caregivers. Inappetence, lack of activity, panting in a species classically designed to be nose breathers, decreased interest in surroundings, different activity patterns, and abnormal postures are just a few signs of chronic pain in cats and dogs. Cats are a species that in particular are exemplary in their abilities to hide chronic pain. They will exhibit marked familial withdrawal, finding secluded areas where they may remain for days to weeks when they experience acute and chronic pain.

ACUTE PAIN MANAGEMENT FOR EMERGENT, CRITICAL/INTENSIVE CARE AND TRAUMA PATIENTS

When deciding on a pain management protocol for a patient, always perform a thorough physical examination and include a pain score assessment before injury and pain has occurred, whenever possible. Form a problem list to guide your choice of anesthesia and analgesia. For example, using a nonsteroidal antiinflammatory drug (NSAID) in an animal with renal failure would not be wise. Remember to account for current medications that the patient may be taking that may augment or interfere with the analgesic or anesthetic drugs. Use multimodal techniques and regional therapy and drugs to target pain at different sites before it occurs. Once a strategy is decided upon, frequently reassess the patient and tailor the protocol to meet each patient's response and needs.

METHODS TO REDUCE PAIN

Drug therapy (in particular, opioids with or without α_2 -agonists) is a cornerstone for acute pain treatment and surgical preemptive pain prevention. However, local anesthetics delivered epidurally, via perineural or plexus injection, intraarticular or trigger point injection, are also effective analgesics for acute and chronic forms of pain and inflammation. The NSAIDs that classically have been reserved for treatment of more chronic or persistent pain states now are being used regularly for treatment of acute and perioperative pain once blood pressure, coagulation, and gastrointestinal parameters have been normalized.

PHARMACOLOGIC MEANS TO ANALGESIA: MAJOR ANALGESICS

OPIOIDS

An opioid is any natural or synthetic drug that is derived from the poppy, which interacts with opiate receptors identified on cell membranes. The drugs from this class constitute the most effective means of controlling acute, perioperative, and chronic pain in human and veterinary medicine (Table 1-16). Their physiologic effects result from the interaction with one or more of at least five endogenous opioid receptors (μ , σ , δ , ϵ , and κ). μ -Receptor agonists are noted for their ability to produce profound analgesia with mild sedation. These drugs diminish “wind-up,” the hyperexcitable state resulting from an afferent volley of nociceptive impulses. They elevate the pain threshold and are used preemptively to prevent acute pain.

As a class, opioids cause CNS depression with their intense analgesia. Dose-related respiratory depression reflects diminished response to carbon dioxide levels. Cardiac depression is secondary only to bradycardia and is more likely with certain opioids such as morphine and oxymorphone. Narcotics produce few if any clinically significant cardiovascular effects in dogs and cats; they are considered cardiac soothing or sparing. Because opioids increase intracranial and intraocular pressure, use them more cautiously in patients with severe cranial trauma and or ocular lesions. Opioids directly stimulate the chemoreceptor trigger zone and may cause nausea and vomiting. Most opioids depress the cough reflex via a central mechanism; this may be helpful in patients recovering from endotracheal intubation irritation. A key characteristic of opioids that makes them desirable for use in emergency and critical care situations is their reversibility. Antagonists block or

TABLE 1-16 Drugs Used in Pain Management

Drug	Agonist effects	Dose	Cardiovascular effects	Disadvantage/Side effects
Fentanyl	Pure μ	2 $\mu\text{g/kg}$ IV bolus 2-8 $\mu\text{g/kg/hour}$ CRI 10-20 $\mu\text{g/kg/hour}$ CRI (inotropic)	Minimal	Hypoventilation at high doses
Buprenorphine	Partial agonist	0.005-0.03 mg/kg q8h IM, IV, SQ; can be placed topically on oral mucosa in cats	Minimal	Partial agonist activity, so not as potent as pure μ -agonists
Butorphanol	Agonist/antagonist	0.2-1.0 mg/kg q2-4h IM, IV, SQ	Minimal	Poor analgesic, adequate sedative if used in combination with an anxiolytic; extremely short duration of action; ceiling effect—more is not better
Codeine	Pure agonist	1-4 mg/kg PO q6h (dogs)	Minimal	Constipation, dysphoria
Morphine	Pure agonist	0.1-0.5 mg/kg q4-8h IM, IV, SQ 0.05-0.1 mg/kg/hour IV CRI	Minimal, can cause histamine release and hypotension if IV is high dose	
Oxymorphone	Pure agonist	0.02-0.1 mg/kg q4-12h IM, IV, SQ	Minimal	Noise hypersensitivity, dysphoria, panting when given IV
Hydromorphone	Pure agonist	0.02-0.2 mg/kg q4-12h IM, IV, SQ	Minimal	Panting during IV, vomiting; hyperthermia in cats

reverse the effect of agonists by combining with receptors and producing minimal or no effects. Administer all reversal agents, such as naloxone and naltrexone, slowly if given intravenously and to effect.

α_2 -AGONISTS

As a class of drugs, α_2 -agonists warrant special attention because most members of the group possess potent analgesic power at doses that are capable of causing sedation, CNS depression, cardiovascular depression, and even general anesthetic states. Originally developed for antihypertensive use, α_2 -agonists quickly have attained sedative analgesic status in veterinary medicine (Table 1-17). Like the opioids, α_2 -agonists produce their effects by aggravating α -adrenergic receptors in the CNS and periphery.

TABLE 1 - 17 α_2 -Agonists Used for Analgesia and Sedation			
Drug	Dose	Effects	Proposed uses
Xylazine	0.05-0.1 mg/kg IV	Short duration	Microdose to decrease dysphoria and anxiety
		Profound cardiovascular depression	
Xylazine		Vomiting, chemoreceptor trigger zone stimulation	Short-duration procedures in HEALTHY dogs
		Bradycardia, vasoconstriction	
		Second-degree atrioventricular block	
		Reversible with yohimbine	
Medetomidine	0.001-0.005 mg/kg IV, IM q4-6h (dog)	Longer duration	Microdose to decrease dysphoria and anxiety
	0.01-0.03 mg/kg IV, IM q4-6h (cat)	Cardiovascular depression	
		Vasoconstriction	Augment analgesia in orthopedic procedures
		Bradycardia, second-degree atrioventricular block	
Medetomidine	1-3 μ g/kg/hour	Reversible with atipamezole (Antesedan)	Short-duration procedures in HEALTHY dogs
		Vomiting, chemoreceptor trigger zone stimulation	

NONSTEROIDAL ANTIINFLAMMATORY DRUGS

Nonsteroidal antiinflammatory drugs, which classically have been used to treat chronic pain and inflammation, as well as cardiovascular diseases, have taken on a new role in the treatment of perioperative and acute pain. Recently, the development of potent oral and parenteral forms of these drugs has compared favorably with and sometimes superiorly to the use of opioids for treatment of acute inflammation and pain (Table 1-18). Nonsteroidal drugs can be used alone, but their best use is that of providing synergistic analgesia with different classes of analgesics (narcotics) or modalities (local, regional and epidural analgesia, physical therapy, acupuncture).

Most NSAIDS act by cyclooxygenase (aka prostaglandin synthetase) inhibition, an enzyme that catalyzes the incorporation of molecular oxygen into arachidonic acid to produce mediators of inflammation. There are at least a few forms of cyclooxygenase,

TABLE 1 - 18 Nonsteroidal Antiinflammatory Drugs and Dosages	
Drug	Dose
Carprofen	2-4 mg/kg PO, IM, SQ, IV q12-24h
Etodolac (EtoGesic)	10-15 mg/kg q24h (dogs only)
Ketoprofen (cat)	0.5-1.0 mg/kg IM, SQ, IV, PO q12h (dogs); q48-72h (cats)
Meloxicam	0.1-0.2 mg/kg PO q24h (dogs); q48-72h (cats)
Piroxicam	0.3 mg/kg PO q48h (dogs and cats)
Ketorolac	0.25-0.5 mg/kg IM, SQ, IV q12h (dogs only)
Deracoxib (Deramaxx)	3-4 mg/kg PO q24h (dogs)
Acetaminophen	10-15 mg/kg PO q6-8h (dogs only)
Aspirin (dog)	10 mg/kg PO q12h
Aspirin (cat)	10 mg/kg PO q48-72h

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among them cyclooxygenase-1 (COX-1), the major constitutive enzyme primarily involved in normal physiologic functions, and COX-2, the enzyme responsible for most of the hyperalgesia and pain responses experienced after tissue injury or trauma. Some NSAIDs inhibit cyclooxygenase and lipoxygenase activity. Most of the currently available oral and parenteral NSAIDs for small animal medicine and surgery target the cyclooxygenase pathways predominantly, although one (tepoxalin) is thought to inhibit both pathways. Inhibition of COX-1 and COX-2 can inhibit the protective effects and impair platelet aggregation and lead to gastrointestinal ulceration.

There are definite contraindications and relative contraindications for the use of NSAIDs. Nonsteroidal antiinflammatory drugs should not be administered to patients with renal or hepatic insufficiency, dehydration, hypotension or conditions that are associated with low circulating volume (congestive heart failure, *unregulated anesthesia*, shock), or evidence of ulcerative gastrointestinal disease. TRAUMA PATIENTS should be stabilized completely regarding vascular volume, tone, and pressure before the use of NSAIDs. Patients receiving concurrent administration of other NSAIDs or corticosteroids, or those considered to be cushingoid, should be evaluated carefully for an adequate “washout” period (time of clearance of drug from the system) before use of an NSAID or before switching NSAIDs. Patients with coagulopathies, particularly those that are caused by platelet number or function defects or those caused by factor deficiencies, and patients with severe, uncontrolled asthma or other bronchial disease are probably not the patients in which to use NSAIDs. Other advice is that NSAIDs not be administered to pregnant patients or to females attempting to become pregnant because COX-2 induction is necessary for ovulation and subsequent implantation of the embryo. The administration of NSAIDs should be considered **ONLY** in the well-hydrated, normotensive dog or cat with normal renal or hepatic function, with no hemostatic abnormalities, and no concurrent steroid administration.

Nonsteroidal antiinflammatory drugs can be used in many settings of acute and chronic pain and inflammation. Among these are the use in well-stabilized musculoskeletal trauma and surgical pain, osteoarthritis management, meningitis, mastitis, animal bite and other wound healing, mammary or transitional cell carcinoma, epithelial (dental, oral, urethral) inflammation, ophthalmologic procedures, and dermatologic or otic disease. Whereas opioids seem to have an immediate analgesic effect when administered, most NSAIDs will take up to 30 minutes for their effect to be recognized. As such, most perioperative or acute NSAIDs use is PART of a balanced pain management scheme, one that uses narcotics and local anesthetic techniques. Nonsteroidal antiinflammatory drugs are devoid of many of the side effects of narcotic administration; namely, decreased gastrointestinal motility, altered sensorium, nausea/vomition, and sedation. Nonsteroidal antiinflammatory drugs are also devoid of many of the side effects of steroid administration; namely, suppression of the pituitary adrenal axis.

Nonsteroidal antiinflammatory drugs in cats

The toxic effects of salicylates in cats are well documented. Cats are susceptible because of slow clearance and dose-dependent elimination because of deficient glucuronidation in this species. Because of this, the dose and the dosing interval of most commonly used NSAIDs need to be altered in order for these drugs to be used. Cats that have been given canine doses of NSAIDs (twice daily or even once daily repetitively) may show hyperthermia, hemorrhagic or ulcerative gastritis, kidney and liver injury, hyperthermia, respiratory alkalosis, and metabolic acidosis. Acute and chronic toxicities of NSAIDs have been reported in cats, especially after repeat once daily dosing. Ketoprofen, flunixin, aspirin, carprofen, and meloxicam have been administered safely to cats, although like most antibiotics and other medications, they are not approved and licensed for use in cats. An important note, though, is that dosing intervals ranging from 48 to 96 hours have been used, and antithrombotic effects often can be achieved at much lower doses than those required to treat fevers and inflammation. I recommend the use of no loading doses, minimum 48-hour dosing intervals, and assurance of adequate circulating blood volume, blood pressure, and renal function.

Because many of the NSAIDs are used off-label in cats, it is imperative that the clinician carefully calculate the dose, modify the dosing interval, and communicate this information to the client before dispensing the drug. Even drugs that come in liquid form (meloxicam), if administered to cats via box-labeled directions used for dogs, will be given in near toxic doses. To worsen the misunderstanding about dosages for cats, drops from manufacturer's bottles often are calibrated drops; when these same liquids are transferred into pharmacy syringes for drop administration, the calibration of course is lost, and the animal potentially is overdosed. A more accurate method of dispensing and administering oral NSAIDs in cats is to calculate the dose in milligrams and determine the exact number of milliliters to administer, rather than use the drop method.

ANALGESIA: MINOR ANALGESICS

Ketamine classically was considered a dissociative anesthetic, but it also has potent activity as an *N*-methyl-D-aspartate (NMDA) receptor antagonist. This receptor located in the CNS mediates windup and central sensitization (a pathway from acute to chronic pain). Blockade of this receptor with microdoses of ketamine results in the ability to provide body surface, somatic, and skin analgesia with potentially lower doses of opioids and α -agonists. Loading doses of 0.5 to 2 mg/kg are used intravenously with continuous rate infusions of 2 to 20 μ g/kg/minute. In and of itself, this drug possesses little to no analgesic ability and indeed in high doses alone often can aggravate, sensitize, or excite the animal in subacute or acute pain.

Amantadine is another NMDA blocker that has been used for its antiviral and Parkinson's stabilizing effects. Amantadine has been used for neuropathic pain in human beings but is only available in an oral form. Suggested starting doses for cats and dogs range from 3 to 10 mg/kg PO daily. When the drug is given orally and intravenously, patients are unlikely to develop behavioral or cardiorespiratory effects with ketamine or amantadine.

Tramadol is an analgesic that possesses weak opioid μ -agonist activity and norepinephrine and serotonin reuptake inhibition. Tramadol is useful for mild to moderate pain in small animals. Although the parent compound has very weak opioid activity, the metabolites have excellent binding affinity for the μ -receptor. Tramadol has been used for peri-surgical pain control when given orally in cats and dogs at a dose of 1 to 10 mg/kg PO sid to bid. Cats appear to require only once daily dosing. Regardless of its affinity for the opioid receptors, the true mechanism of action of tramadol in companion animals remains largely unknown.

Gabapentin is a synthetic analog of γ -aminobutyric acid (GABA). Originally introduced as an antiepileptic drug, the mechanism of action of gabapentin remains somewhat unclear in veterinary medicine. The drug is among a number of commonly used antiepileptic medications used to treat central pain in human beings. The rationale for use is the ability of the drugs to suppress discharge in pathologically altered neurons. Gabapentin does this through calcium channel modulation without binding to glutamate receptors. Chronic, burning, neuropathic, and lancinating pain in small animals responds well to 1 to 10 mg/kg PO daily.

ADJUNCTIVE ANALGESIC DRUGS

Local anesthetic agents are the major class used as a peripheral-acting analgesic (Table 1-19). Local anesthetics block the transmission of pain impulses at the peripheral nerve nociceptor regions. Local anesthetics may be used to block peripheral nerves or inhibit nerve "zones" using regional techniques. Although all local anesthetics are capable of providing pain relief, agents with a longer duration of action are preferred for pain management purposes. Bupivacaine is an example of a long-acting local anesthetic drug that is used along with lidocaine for long-acting pain relief. A single dose of bupivacaine injected at a local site will provide local anesthesia and analgesia for 6 to 10 hours.

TABLE 1-19 Analgesics		
Drug	Dose	Use
Amantadine	3 mg/kg, PO q24h (dogs and cats)	Chronic pain
Dextromethorphan	1-2 mg/kg PO q6-8h (dogs and cats)	Prevent windup
Gabapentin	1.25-10 mg/kg PO q24h (dogs and cats)	Chronic pain
Tramadol	1-10 mg/kg PO q8-24h	Acute and chronic pain

TABLE 1-20 Commonly Used Analgesic Assistance Drugs		
Drug	Dose	
Acepromazine	0.01-0.03 mg/kg IV, IM, SQ q8-24 hours	0.2-0.5 mg/kg PO q12-24h
Diazepam	0.5-1.0 mg/kg IV in cats and dogs, followed by 0.1-0.2 mg/kg/hour IV CRI	
Midazolam	0.3-0.5 mg/kg IV, IM, SQ in cats and dogs, followed by 0.05 mg/kg/hour IV CRI	

Combination Approach: Mix with one another and give as a constant rate infusion at 10 mL/kg/hour

Drug	Dose	CRI dose provided
Morphine	5 mg in 500 mL	0.1 mg/kg/hour
Lidocaine	150 mg	3 mg/kg/hour
Ketamine	100 mg	2 mg/kg/hour

When lidocaine is administered as an intravenous constant rate infusion (50 to 75 µg/kg/minute in dogs, 1 to 10 µg/kg/minute in cats) is effective in the treatment of chronic neuropathic pain and periosteal and peritoneal pain (e.g., pancreatitis). Mexiletine, an oral sodium channel blocker, can be used as an alternative to injectable lidocaine for provision of background analgesia.

ANXIOLYTICS AND SEDATIVES

Many drugs (Table 1-20) are used in combination with opioids, α₂-agonists, and ketamine to provide anxiolysis and sedation.

LOCAL AND REGIONAL TECHNIQUES FOR THE EMERGENT PATIENT

Injection of local anesthetic solution into the connective tissue surrounding a particular nerve produces loss of sensation (sensory blockade) and/or paralysis (motor nerve blockade) in the region supplied by the nerve. Local anesthetics also may be administered epidurally, intrathoracically, intraperitoneally, and intraarticularly. Lidocaine and bupivacaine are the most commonly administered local anesthetics. Lidocaine provides for quick, short-acting sensory and motor impairment. Bupivacaine provides for later-onset, longer-lasting desensitization without motor impairment. Combinations of the two agents diluted with saline are used frequently to provide for quick-onset analgesia that lasts between 4 and 6 hours in most patients. Adding narcotic and/or α₂ agent often maximizes the analgesia and increases the pain-free interval to 8 to 18 hours. Epinephrine and preservative-free solutions are recommended. Precision placement of anesthetic close to nerves, roots, or plexuses is improved with the use of a stimulating nerve locator. Cats seem to be more

sensitive to the effects of local anesthetics; as such the lower ends of most dosing ranges are used for blockades in this species.

Unlike most instances of general anesthesia, during which the animal is rendered unconscious and nerve transmission is decreased by virtue of CNS depression, local and regional techniques block the initiation of noxious signals, thereby effectively preventing pain from entering the CNS. This is an effective means of not only preventing initial pain but also reducing the changes that take place in the dorsal horn of the spinal cord, spinothalamic tracts, limbic and reticular activating centers, and cortex. Frequently, the neurohormonal response that is stimulated in pain and stress is blunted as well. Overall, the patient has fewer local and systemic adverse effects of pain, disease processes are minimized, chronic pain states are unlikely, and outcome is improved. Regional techniques are best used as part of an analgesic regimen that consists of their continuous administration, narcotics, α -agonists, anxiolytics, and good nursing.

TOPICAL AND INFILTRATIVE BLOCKADE

Lidocaine can be added to sterile lubricant in a one-to-one concentration to provide decreased sensation for urinary catheterization, nasal catheter insertion, minor road burn analgesia, and pyotraumatic dermatitis analgesia. Proparacaine is a topical anesthetic useful for corneal or scleral injuries. Local anesthetics can be used to infiltrate areas of damage or surgery by using long-term continuous drainage catheters and small, portable infusion pumps. This is an effective means of providing days of analgesia for massive surgical or traumatic soft tissue injury. Even without the catheter, incisional or regional soft tissue blocking using a combination of 1 to 2 mg/kg lidocaine and 0.5 to 2 mg/kg bupivacaine diluted with equal volume of saline and 1:9 with sodium bicarbonate is effective for infiltrating large areas of injury.

CRANIAL NERVE BLOCKADE

Administration of local anesthetic drugs around the infraorbital, maxillary, ophthalmic mental, and alveolar nerves can provide excellent analgesia for dental, orofacial, and ophthalmic trauma and surgical procedures. Each nerve may be desensitized by injecting 0.1 to 0.3 mL of a 2% lidocaine hydrochloride solution and 0.1 to 0.3 mL of 0.5% bupivacaine solution using a 1.2- to 2.5-cm, 22- to 25-gauge needle. Precise placement perineurally versus intraneurally (neuroma formation common) is enhanced by using catheters in the foramen versus needle administration. Always perform aspiration before administration to rule out intravascular injection of agents.

INTRAPLEURAL BLOCKADE

This block is used to provide analgesia for thoracic, lower cervical, cranial abdominal, and diaphragmatic pain. Following aseptic preparation, place a small through-the-needle (20- to 22-gauge) catheter in the thoracic cavity between the seventh and ninth intercostal space on the midlateral aspect of the thorax. Aseptically mix a 0.5 to 1 mg/kg lidocaine and a 0.2 to 0.5 mg/kg bupivacaine dose with volume of saline equal to the volume of bupivacaine, and slowly inject it over a period of 2 to 5 minutes following aspiration to ensure that no intravascular injection occurs. Depending on where the lesion is, position the patient to allow the intrapleural infusion to “coat” the area. Most effective is positioning the patient in dorsal recumbency for several minutes following the block to make sure local anesthetic occupies the paravertebral gutters and hence the spinal nerve roots. The block should be repeated every 3 hours in dogs and every 8 to 12 hours in cats. Secure the catheter to the skin surface for repetitive administration.

BRACHIAL PLEXUS BLOCKADE

Administration of local anesthetic around the brachial plexus provides excellent analgesia for forelimb surgery, particularly that distal to the shoulder, and amputations. Nerve locator-guided techniques are much more accurate and successful than blind placement of local anesthetic; however, even the latter is useful.

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- To administer a brachial plexus blockade, follow this procedure:
1. Aseptically prepare a small area of skin over the point of the shoulder.
 2. Insert a 22-gauge, 1½- to 3-inch spinal needle medial to the shoulder joint, axial to the lesser tubercle, and advance it caudally, medial to the body of the scapula, and toward the costochondral junction of the first rib. Aspirate first before injection to make sure that intravenous injection does not occur.
 3. Inject one third of the volume of local anesthetic mix, and then slowly withdraw the needle and fan dorsally and ventrally while infusing the remaining fluid.
 4. Local anesthetic doses are similar to those for intrapleural blockade.

EPIDURAL ANESTHESIA AND ANALGESIA

Epidural analgesia refers to the injection of an opioid, a phencyclidine, an α -agonist, or an NSAID into the epidural space. Epidural anesthesia refers to the injection of a local anesthetic. In most patients a combination of the two is used. Epidural analgesia and anesthesia are used for a variety of acute and chronic surgical pain or traumatically induced pain in the pelvis, tail, perineum, hind limbs, abdomen, and thorax (Table 1-21). Procedures in which epidural analgesia and anesthesia are useful include forelimb and hind limb amputation, tail or perineal procedures, cesarean sections, diaphragmatic hernia repair, pancreatitis, peritonitis, and intervertebral disk disease. Epidural blocks performed using opioids or bupivacaine will not result in hind limb paresis or decreased urinary or anal tone (incontinence), unlike lidocaine or mepivacaine epidural blocks. Morphine is one of the most useful opioids for administration in the epidural space because of its slow systemic absorption. Epidural catheters used for the instillation of drugs through constant rate infusion or intermittent injection can be placed in dogs and cats. Routinely placed at the lumbosacral junction, these catheters are used with cocktails including preservative-free morphine, bupivacaine, medetomidine, and ketamine. Extremely effective for preventing windup pain in the peritoneal cavity or caudal half of the body, the catheters may be maintained if placed aseptically for 7 to 14 days.

- To provide epidural analgesia or anesthesia, follow this procedure:
1. Position the animal in lateral or sternal recumbency.
 2. Clip and aseptically scrub over the lumbosacral site.
 3. Palpate the craniodorsal-most extent of the wings of the ileum bilaterally and draw an imaginary line through them to envision the spine of L7 located immediately behind the imaginary line.
 4. Advance a 20- to 22-gauge, 1½- to 3-inch spinal or epidural needle through the skin just caudal to the spine of L7.
 5. The needle will lose resistance as it is introduced into the epidural space. Drop saline into the hub of the needle, and the saline will be pulled into the epidural space as the needle enters.

TABLE 1-21 Drugs to Use for Epidural Anesthesia	
Drug	Dose
Bupivacaine 0.25%*	0.1-0.3 mg/kg (1 mL/5 kg epidural q4-6h (canine only, not recommended for cats)
Morphine (Duramorph)*,†	0.05-0.1 mg/kg spinal q8h

*Preservative-free solutions should be used, with filtered needles or in-line filters if an epidural catheter with constant rate infusion is used.
†Can be diluted to a total volume of 0.1 to 0.15 mL/kg with sterile saline if advancement of the solution into the thoracic area is desired (forelimb amputation, thoracostomy, diaphragmatic hernia repair).

INTERCOSTAL NERVE BLOCKS

Discrete intercostal nerve blocks can provide effective analgesia for traumatic or postsurgical pain. Identify the area of the injury, and infiltrate three segments on either side of the injury with analgesic.

To perform an intercostal nerve block, follow this procedure:

1. Clip and aseptically scrub the dorsal and ventral third of the chest wall.
2. Palpate the intercostal space as far dorsally as possible.
3. Use a 25-gauge, 0.625-inch needle at the caudolateral aspect of the affected rib segments and those cranial and caudal.
4. Direct the tip of the needle caudally such that the tip of the needle “drops” off of the caudal rib. (This places the needle tip in proximity to the neuromuscular bundle that contains the intercostal nerve that runs in a groove on the caudomedial surface of the rib.)
5. Aspirate to confirm that the drug will not go intravenously.
6. Inject while slowly withdrawing the needle. Inject 0.5 to 1.0 mL at each site, depending on the size of the animal.

Additional Reading

Gaynor JS, Muir WW: *Handbook of veterinary pain management*, St Louis, 2003, Mosby, 2003.
Melzack R, Wall PD: *Handbook of pain management, a clinical companion to Wall and Melzack's textbook of pain*, Edinburgh, 2003, Churchill Livingstone.
Muir WW, Hubbell JAE, Skarda RT, et al: *Handbook of veterinary anesthesia*, ed 3, St Louis, 2000, Mosby.

EMERGENCY MANAGEMENT OF SPECIFIC CONDITIONS

ACUTE CONDITION IN THE ABDOMEN

An acute condition in the abdomen is defined as the sudden onset of abdominal discomfort or pain caused by a variety of conditions involving intraabdominal organs. Many animals have the primary complaint of lethargy, anorexia, ptyalism, vomiting, retching, diarrhea, hematochezia, crying out, moaning, or abnormal postures. Abnormal postures can include generalized rigidity, walking tenderly or as if “on eggshells,” or a prayer position in which the front limbs are lowered to the ground while the hind end remains standing. In some cases, it may be difficult initially to distinguish between true abdominal pain or referred pain from intervertebral disk disease. Rapid progression and decompensation of the patient's cardiovascular status can lead to stupor, coma, and death in the most extreme cases, making rapid assessment, treatment, and definitive care extremely challenging.

SIGNALMENT AND HISTORY

Often the patient's signalment and history can increase the index of suspicion for a particular disease process. A thorough history often is overlooked or postponed in the initial stages of resuscitation of the patient with acute abdominal pain. Often, asking the same question in a variety of methods can elicit an answer from the client that may lead to the source of the problem and the reason for acute abdominal pain. Important questions to ask the client include the following:

- What is your chief complaint or reason that you brought your animal in on emergency?
- When did the signs first start, or when was your animal last normal?
- Do you think that the signs have been the same, better, or getting worse?
- Does your animal have any ongoing or past medical problems?
- Have similar signs occurred in the past?
- Does your animal have access to any known toxins, or does he or she run loose unattended?

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- Has your animal ingested any garbage, compost, or table scraps recently?
- Are there any other animals in your household, and are they acting sick or normal?
- Has your animal been vaccinated recently?
- Has there been any change in your pet's appetite?
- Have you noticed any weight loss or weight gain?
- Have you noticed any increase or decrease in water consumption or urination?
- Does your animal chew on bones or toys?
- Have you noticed any toys, socks, underwear, or other items missing from your household?
- Is there a possibility of any trauma including being hit by a car or kicked by a larger animal or person?
- Have you noticed a change in your pet's defecation habits?
- Have you seen any vomiting or diarrhea?
- What does the vomitus or diarrhea look like?
- Is the vomitus in relation to eating?
- Is there any blood or mucus in the vomitus or diarrhea?
- When was the last time your animal vomited or had diarrhea?
- When your animal vomits, does it actively retch with abdominal contractions, or is it more passive like regurgitation?
- What is the color of the feces? Is it black or red?
- Does the vomit smell malodorous like feces?

IMMEDIATE ACTION

As with any other emergency, the clinician must follow the ABCs of therapy, treating the most life-threatening problems first. First, perform a perfunctory physical examination. Examination of the abdomen ideally should be performed last, in case inciting a painful stimulus precludes you from evaluating other organ systems more thoroughly. Briefly observe the patient from a distance. Are there any abnormal postures? Is there respiratory distress? Is the animal ambulatory, and if so, do you observe any gait abnormalities? Do you observe any ptialism or attempts to vomit? Auscultate the patient's thorax for crackles that may signify aspiration pneumonia resulting from vomiting. Examine the patient's mucous membrane color and capillary refill time, heart rate, heart rhythm, and pulse quality. Many patients in pain have tachycardia that may or may not be accompanied by dysrhythmias. If a patient's heart rate is inappropriately bradycardic, consider hypoadrenocorticism, whipworm infestation, or urinary obstruction or trauma as a cause of hyperkalemia. Assess the patient's hydration status by evaluating skin turgor, mucous membrane dryness, and whether the eyes appear sunken in their orbits. A brief neurologic examination should consist of whether the patient is actively having a seizure, or whether mental dullness, stupor, coma, or nystagmus are present. Posture and spinal reflexes can assist in making a diagnosis of intervertebral disk disease versus abdominal pain. Perform a rectal examination to evaluate for the presence of hematochezia or melena.

Finally, examination of the abdomen should proceed first with superficial and then deeper palpation. Visually inspect the abdomen for the presence of external masses, bruising, or penetrating injuries. Reddish discoloration of the periumbilical area often is associated with the presence of intraabdominal hemorrhage. It may be necessary to shave the fur to inspect the skin and underlying structures visually for bruising and ecchymoses. Auscultate the abdomen for the presence or absence of borborygmi to characterize gut sounds. Next, perform percussion and ballottement to evaluate for the presence of a gas-distended viscus or peritoneal effusion. Finally, perform first superficial and then deep palpation of all quadrants of the abdomen, noting abnormal enlargement, masses, or whether focal pain is elicited in any one area. Once the physical examination has been performed, implement initial therapy in the form of analgesia, fluid resuscitation, and antibiotics.

TREATMENT

Treatment for any patient with an acute condition in the abdomen and shock is to treat the underlying cause, maintain tissue oxygen delivery, and prevent end-organ damage and failure. A more complete description of shock and oxygen delivery is given in the section on shock.

ANALGESIA

The administration of analgesic agents to any patient with acute abdominal pain is one of the most important therapies in the initial stages of case management. Table 1-22 lists analgesic drugs for use in the patient with an acute condition in abdomen. Table 1-23 lists analgesic and anxiolytic drugs to avoid in the patient with an acute condition in abdomen.

TABLE 1-22 Analgesic Agents for Use in Dogs and Cats with Acute Abdominal Pain

Drug	Dose
Butorphanol	0.1-0.2 mg/kg IV (dogs and cats); 0.2-0.4 mg/kg SQ or IM (dogs and cats)
Buprenorphine	0.005-0.02 mg/kg IV, IM, SQ q6-12h (dogs) 0.005-0.01 mg/kg IV, IM, SQ, q6-12h (cats) (Also can be placed in the mouth for buccal absorption in cats)
Fentanyl	2 µg/kg IV bolus, followed by 3-7 µg/kg/hour CRI (dogs and cats)
Hydromorphone	0.1-0.2 mg/kg SQ, IM, IV (dogs and cats)
Lidocaine	1-2 mg/kg IV slowly over 2-5 minutes, then 30-50 µg/kg/minute CRI
Morphine	0.5-1.0 mg/kg SQ, IM; 0.1 mg/kg/hour CRI (dogs) 0.25-0.5 mg/kg SQ, IM; 0.05 mg/kg/hour CRI (cats)

TABLE 1-23 Analgesic and Anxiolytic Agents That Are Contraindicated and Should Be Avoided in the Patient with Acute Abdominal Pain

Drug	Potential risks
<i>α-Antagonists</i> Acepromazine Chlorpromazine	α-Receptor antagonist, hypotension
<i>α₂-Agonists</i> Xylazine Medetomidine	α ₂ -agonist, peripheral vasoconstriction, dose-dependent decrease in cardiac output, hypotension
<i>Antiprostaglandins</i> Aspirin Flunixin meglumine Indomethacin Phenylbutazone Ibuprofen Carprofen Ketoprofen Aminopyrine Flufenamic acid	Decreased renal and gastrointestinal perfusion, gastrointestinal ulceration
<i>Glucocorticoids</i> Dexamethasone Dexamethasone sodium phosphate Hydrocortisone sodium phosphate Prednisone Prednisolone sodium phosphate Methylprednisolone sodium succinate	Decreased renal and gastrointestinal perfusion, gastrointestinal ulceration

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FLUID RESUSCITATION

Many patients with acute abdominal pain are clinically dehydrated or are in hypovolemic shock because of hemorrhage. Careful titration of intravenous crystalloid and colloid fluids including blood products is necessary based on the patient's perfusion parameters including heart rate, capillary refill time, blood pressure, urine output, and PCV. Fluid therapy also should be based on the most likely differential diagnoses, with specific fluid types administered according to the primary disease process. In dogs, a shock volume of fluids is calculated based on the total blood volume of 90 mL/kg/hour. In cats, shock fluid rate is based on plasma volume of 44 mL/kg/hour. In most cases, any crystalloid fluid can be administered at an initial volume of one fourth of a calculated shock dose and then titrated according to whether the patient's cardiovascular status responds favorably or not. In cases of an acute condition in the abdomen from known or suspected hypoadrenocorticism, severe whipworm infestation, or urinary tract obstruction or rupture, 0.9% sodium chloride fluid without added potassium is the fluid of choice. When hemorrhage is present, the administration of whole blood or packed RBCs may be indicated if the patient has clinical signs of anemia and shows clinical signs of lethargy, tachypnea, and weakness. Fresh frozen plasma is indicated in cases of hemorrhage resulting from vitamin K antagonist rodenticide intoxication or hepatic failure or in cases of suspected disseminated intravascular coagulation (DIC). A more thorough description of fluid therapy is given under the sections on shock and fluid therapy.

ANTIBIOTICS

The empiric use of broad-spectrum antibiotics is warranted in cases of suspected sepsis or peritonitis as a cause of acute abdominal pain. Ampicillin sulbactam (22 mg/kg IV q6-8h) and enrofloxacin (10 mg/kg once daily) are the combination treatment of choice to cover gram-negative, gram-positive, aerobic, and anaerobic infections. Alternative therapies include a second-generation cephalosporin such as cefotetan (30 mg/kg IV tid) or cefoxitin (22 mg/kg IV tid) or added anaerobic coverage with metronidazole (10 to 20 mg/kg IV tid).

OXYGEN SUPPLEMENTATION

Tissue oxygen delivery depends on a number of factors, including arterial oxygen content and cardiac output. If an animal has had vomiting and subsequent aspiration pneumonitis, treatment of hypoxemia with supplemental oxygen in the form of nasal, nasopharyngeal, hood, or transtracheal oxygen administration is important (see Oxygen Supplementation under Emergency Diagnostic and Therapeutic Procedures).

DIAGNOSTIC PROCEDURES**Complete blood count**

Perform a complete blood count in all cases of acute abdominal pain to determine if life-threatening infection or coagulopathy including DIC is present. In cases of sepsis, infection, or severe nonseptic inflammation, the white blood cell count may be normal, elevated, or low. Examine a peripheral blood smear for the presence of toxic neutrophils, eosinophils, atypical lymphocytes, nucleated RBCs, platelet estimate, anisocytosis, and blood parasites. A falling PCV in the face of RBC transfusion suggests ongoing hemorrhage.

Biochemistry panel

Perform a biochemistry panel to evaluate organ system function. Azotemia with elevated BUN and creatinine may be associated with prerenal dehydration, impaired renal function, or postrenal obstruction or leakage. The BUN also can be elevated when gastrointestinal hemorrhage is present. Serum amylase may be elevated with decreased renal function or in cases of pancreatitis. A normal serum amylase, however, does not rule out pancreatitis as a source of abdominal pain. Serum lipase may be elevated with gastrointestinal inflammation or pancreatitis. Like amylase, a normal serum lipase does not rule out pancreatitis. Total bilirubin, alkaline phosphatase, and alanine transaminase may be elevated with

primary cholestatic or hepatocellular diseases or may be due to extrahepatic causes including sepsis.

Urinalysis

Obtain a urinalysis via cystocentesis whenever possible, except in cases of suspected pyometra or transitional cell carcinoma. Azotemia in the presence of a nonconcentrated (isosthenuric or hyposthenuric) urine suggests primary renal disease. Secondary causes of apparent renal azotemia and lack of concentrating ability also occur in cases of hypo-adrenocorticism and gram-negative sepsis. Renal tubular casts may be present in cases of acute renal ischemia or toxic insult to the kidneys. Bacteriuria and pyuria may be present with infection and inflammation. When a urinalysis is obtained via free catch or urethral catheterization, the presence of bacteriuria or pyuria also may be associated with pyometra, vaginitis, or prostatitis/prostatic abscess.

Lactate

Serum lactate is a biochemical indicator of decreased organ perfusion, decreased oxygen delivery or extraction, and end-organ anaerobic glycolysis. Elevated serum lactate greater than 6 mmol/L has been associated with increased morbidity and need for gastric resection in cases of GDV and increased patient morbidity and mortality in other disease processes. Rising serum lactate in the face of adequate fluid resuscitation is a negative prognostic sign.

Abdominal radiographs

Obtain abdominal radiographs as one of the first diagnostic tests when deciding whether to pursue medical or surgical management. The presence of GDV, linear foreign body, pneumoperitoneum, pyometra, or splenic torsion warrants immediate surgical intervention. If a loss of abdominal detail occurs because of peritoneal effusion, perform additional diagnostic tests including abdominal paracentesis (abdominocentesis) and abdominal ultrasound to determine the cause of the peritoneal effusion.

Abdominal ultrasound

Abdominal ultrasonography is often useful in place of or in addition to abdominal radiographs. The sensitivity of abdominal ultrasonography is largely operator dependent. Indications for immediate surgical intervention include loss of blood flow to an organ, linear bunching or placcation of the intestinal tract, intussusception, pancreatic phlegmon or abscess, a fluid-filled uterus suggestive of pyometra, gastrointestinal obstruction, intraluminal gastrointestinal foreign body, dilated bile duct, or gallbladder mucocele, or gas within the wall of the stomach or gallbladder (emphysematous cholecystitis). The presence of peritoneal fluid alone does not warrant immediate surgical intervention without cytologic and biochemical evaluation of the fluid present.

Abdominocentesis

See also Abdominal Paracentesis and Diagnostic Peritoneal Lavage.

Abdominal paracentesis (abdominocentesis) often is the deciding factor in whether to perform immediate surgery. Abdominocentesis is a sensitive technique for detecting peritoneal effusion when more than 6 mL/kg of fluid is present within the abdominal cavity. Abdominal effusion collected should be saved for bacterial culture and evaluated biochemically and cytologically based on your index of suspicion of the primary disease process. If creatinine, urea nitrogen (BUN) or potassium is elevated compared with that of serum, uroabdomen is present. Elevated abdominal fluid lipase or amylase compared with serum supports a diagnosis of pancreatitis. Elevated lactate compared with serum lactate or an abdominal fluid glucose less than 50 mg/dL is highly sensitive and specific for bacterial/septic peritonitis. The presence of bile pigment or bacteria is supportive of bile and septic peritonitis, respectively. Free fibers in abdominal fluid along with clinical signs of abdominal pain strongly support gastrointestinal perforation, and immediate surgical exploration is required.

Text continued on p. 93

TABLE 1 - 2.4 Conditions That Can Cause Clinical Signs of Acute Abdominal Pain

The following are clinical conditions, common history, physical examination, and characteristic findings of various diagnostic tests. A blank column next to a condition indicates no specific signalment, history, physical examination, or diagnostic test characteristic for a particular disease process.

Condition	Signalment	History/Chief complaint	Physical examination findings
Abdominal wall			
Hernia	Any	History of trauma, vomiting Abdominal wall swelling Pain, lethargy, anorexia	Abdominal wall swelling, fever, pain
Abscess	Any	Anorexia, pain, lethargy Abdominal wall swelling	Abdominal wall swelling, fever, pain
Blunt trauma	Any	History of trauma, lethargy, pain, inappetence	Pain, hematoma or ecchymosis, periumbilical redness/hemorrhage
Gastrointestinal			
Diaphragmatic hernia	Any	History of trauma, vomiting, lethargy, anorexia, respiratory difficulty	Cyanosis, respiratory difficulty, abdominal pain
Gastroenteritis bacterial	Any	Vomiting, diarrhea, history of toxin or garbage ingestion	Abdominal pain, increased borborygmi vomiting, diarrhea, hematochezia
Parvovirus/Panleukopenia	Young puppy Young kitten	Inadequate vaccination, vomiting, diarrhea, anorexia, lethargy	Dehydration, vomiting, diarrhea, lethargy
Parasitic	Any	Vomiting, diarrhea, history of worms in feces	Ileus, increased or decreased borborygmi
Metabolic/hypoadrenocorticism	Any, young female, specific breed predisposition	Waxing and waning, lethargy, vomiting, diarrhea, weakness, anorexia, weight loss, stress	Muscle atrophy, dehydration, melena, hematochezia, inappropriate bradycardia
Toxin	Any	History of toxin or garbage exposure	Abdominal pain, lethargy
Gastric dilatation	Any	History of garbage or food exposure	Distended abdomen, pyralism
Gastric dilatation-volvulus	Large breed or deep-chested dog; can occur in any breed	History of unproductive retching	Distended painful tympanic abdomen, cyanosis, respiratory difficulty, pyralism, retching or unproductive vomiting

Gastric ulcer	Any	Hematemesis, coffee ground vomitus, lethargy, anorexia, melena	Abdominal pain, melena
Cecal inversion	Any	Vomiting, hematochezia, dyschezia, lethargy	Hematochezia, abdominal pain
Colonic ulcer/perforation	Any	Vomiting, history of exposure to string, thread, ribbon	Abdominal pain, clumped intestines on palpation, string under tongue
Linear foreign body	Any	History of vomiting, inappetence, history of eating foreign object(s)	Abdominal pain, palpate abdominal mass
Luminal foreign body	Any	Vomiting, anorexia, lethargy	Abdominal pain, fever; lethargy, dehydration, palpable mass effect
Intestinal/ulcer perforation	Any	Vomiting, diarrhea, lethargy	Abdominal pain, fever; palpable abdominal mass ("sausage")
Intussusception	Any, primarily young dogs/cats	Vomiting, straining to defecate, crying out in pain, anorexia	Palpable mass effect, dry feces on rectal examination
Obstipation	Older	Vomiting, diarrhea, hematochezia, anorexia, abdominal pain	Abdominal pain, fever; palpable fluid wave, hematochezia with luminal tissue on rectal examination
Vascular ischemia/bowel compromise	Any		
Liver and gallbladder			
Cholangiohepatitis/hepatitis	Any	Anorexia, vomiting, pain, lethargy, icterus	Dehydration, painful abdomen, vomitus, icterus
Cholecystitis/Emphysematous cholecystitis/gallbladder mucocoele	Any	Anorexia, vomiting, pain, lethargy	Dehydration, painful abdomen, vomitus, icterus, fever
Biliary rupture/bile peritonitis	Any	History of trauma, pain, lethargy, vomiting, anorexia	Dehydration, painful abdomen, vomitus, icterus, fever
Biliary obstruction	Any	Anorexia, vomiting, pain, lethargy	Dehydration, painful abdomen, vomitus, fever
Hepatic abscess	Any	Anorexia, vomiting, pain, lethargy	Dehydration, painful abdomen, vomitus, fever
Hepatic torsion	Any	Anorexia, vomiting, pain, lethargy	Painful abdomen, vomitus, fever, dehydration, seizures
Hepatic neoplasia	Any/older animals	Anorexia, vomiting, pain, lethargy	Painful abdomen, vomitus, fever, dehydration, seizures

Continued

TABLE 1-2.4 Conditions That Can Cause Clinical Signs of Acute Abdominal Pain—cont'd

Condition	Signalment	History/Chief complaint	Physical examination findings
Pancreas			
Pancreatitis	Any, some breed Predisposition	Anorexia, vomiting, pain, lethargy History of eating fatty meal	Dehydration, abdominal pain, vomitus, alopecia, palpable abdominal mass
Pancreatic abscess			
Pancreatic pseudocyst or mucocele			
Pancreatic neoplasia	Any, older animals	Anorexia, vomiting, pain, lethargy, weight loss, truncal alopecia	Dehydration, abdominal pain, vomitus, alopecia, palpable abdominal mass
Spleen			
Splenic torsion	Any	Acute pain, vomiting, lethargy	Pale mm, decompensatory shock, palpable abdominal mass and splenomegaly, abdominal pain
Splenic mass	Any/older animals	Acute pain, lethargy, collapse	Pale mm, decompensatory shock, premature ventricular contractions on ECG, anemia
Splenic infarction	Any	Acute pain, lethargy, collapse	Fever, abdominal pain, splenomegaly
Traumatic splenic laceration	Any	History of trauma	Abdominal pain, ballotable fluid wave, anemia, compensatory or decompensatory shock
Genitourinary			
Mastitis	Female	History of lactation	Abdominal pain, fever, lethargy, anorexia painful swollen sometimes abscessed mammary glands, discolored milk
Penis fracture	Male dogs	History of trauma, history of traumatic breeding	Painful abdomen and penis
Paraphimosis	Male dogs	Persistent erection	Swollen penis outside of prepuce
Prostate			
Prostatitis	Male dogs	Straining to defecate	Painful enlarged prostate on rectal palpation, fever
Prostatic abscess	Older male dogs	Straining to defecate, pain, lethargy	Painful enlarged prostate on rectal palpation
Prostatic neoplasia	Older male dogs	Straining to defecate	Enlarged prostate on rectal examination
Renal acute nephritis	Any	Lethargy, vomiting, anorexia	Painful abdomen, dehydration, fever
Pyelonephritis	Any	Lethargy, PU/PD, vomiting, anorexia	Painful abdomen, fever
Renal neoplasia	Any, older	Anorexia, vomiting, lethargy, weight loss	Painful abdomen, fever, cachexia, palpable abdominal mass
Renal abscess	Any	Anorexia, vomiting, lethargy, weight loss	Painful abdomen, fever, cachexia, palpable abdominal mass
Renal infarct/thrombus	Any	Lethargy, PU/PD, vomiting	Painful abdomen, fever

Renolithiasis	Any	Lethargy, PU/PD, vomiting	Painful abdomen, fever
Ureteral obstruction	Any	Lethargy, PU/PD, vomiting	Painful abdomen, fever, dehydration
Ureteral rupture	Any	Lethargy, PU/PD, vomiting	Painful abdomen, fever, dehydration
Urethral obstruction	Any	Lethargy, PU/PD, vomiting, straining to urinate	Painful abdomen, dehydration, vomitus, painful distended nonexpressible urinary bladder
Urethral tear/rupture	Any	Lethargy, PU/PD, vomiting, history of trauma	Painful abdomen, dehydration, vomitus, fever
Urinary bladder neoplasia	Any, older animals	Stranguria, hematuria, weight loss, pollakiuria	Thickened urethra may be palpable on rectal examination
Testicles			
Testicular torsion	Intact male dogs	Pain, chewing or looking at back end	Swollen painful testicle, fever
Uterus and Ovaries			
Uterine torsion	Intact gravid females	Acute collapse, vaginal discharge, history of breeding	Decompensatory shock, vaginal discharge
Pyometra	Intact females	Recent heat cycle, PU/PD, vomiting, diarrhea, lethargy, vaginal discharge	Dehydration, soft tissue mass in caudal abdomen, vaginal discharge, fever
Uterine rupture	Intact gravid females	History of recent whelping/queening, lethargy, acute collapse	Abdominal pain, vaginal discharge, decompensatory shock
Other			
Discospondylitis	Any	History of pain, lethargy, anorexia	Painful spine, fever
Envenomation			
Black widow spider	Any	History of possible exposure, pain, acute collapse, vomiting	Recumbency, muscle fasciculations, pain, vomiting, fever, collapse
Brown recluse	Any	History of possible exposure, pain, necrotizing bulls-eye ulcer formation	Ulcer, pain, fever, granulomatous lesion
Intervertebral disk disease	Any	Acute paresis or paralysis	Paresis or paralysis, spinal pain
Meningitis	Any	Acute pain, lethargy, anorexia	Fever, extreme pain
Myositis	Any	Acute pain, lethargy, anorexia	Fever, extreme pain
Neoplasia	Any	Acute pain, lethargy, anorexia, collapse	Decompensatory shock, palpable abdominal mass
Peritonitis	Any	Vomiting, anorexia, lethargy, pain, history of trauma or penetrating abdominal injury	Pain, fever, palpable foreign object
Sublumbar or retroperitoneal abscess	Any	Pain, anorexia, lethargy	Pain, lethargy, dehydration, fever

Continued

TABLE 1-24 Conditions That Can Cause Clinical Signs of Acute Abdominal Pain—cont'd

Diagnostic tests	Treatment
Lack of contiguity of body wall	Surgical (immediate)
Soft tissue density or abdominal contents SQ on RADS	
Lack of contiguity of body wall	Surgical (immediate)
Soft tissue density or abdominal contents SQ on RADS; inflammatory cells and bacteria on aspirate	
Hemoabdomen on abdominocentesis or DPL	Medical
Radiographic evidence of abdominal organs in thorax, may require contrast celiotomy	Medical unless stomach is in thorax
Ileus on radiographs, white blood cells in feces	Medical
Parvovirus CITE test positive on feces leuko-/neutropenia	Medical
Parasite oocysts or parasites in feces	Medical
Atrial standstill on EGG, hyperkalemia, hyponatremia, hypocholesterolemia, hypoglycemia, hyperphosphatemia, azotemia, normal WBC and differential positive ethylene glycol	Medical
Calcium oxalate dihydrate crystals U/A “halo sign” = hyperechoic renal cortex on U/S	Medical
Soft tissue density/food with gastric dilation on radiographs	Medical
Dorsal and cranial displacement of pylorus with dilation of gastric fundus on right lateral radiograph, premature ventricular contractions on ECG, elevated lactate	Surgical (Immediate)
Regenerative anemia, melena, loss of abdominal detail on radiographs if perforation present	Medical unless perforation present
Loss of detail on radiographs if perforation and peritonitis present	Medical unless perforation present
C-shaped abnormal gas pattern with plication on radiographs	Surgical (Immediate)
Dilation of bowel cranial to foreign object, radiopaque object in stomach or intestines, hypochloremic metabolic acidosis on bloodwork if pyloric outflow obstruction is present	Surgical (Immediate)
Elevated or decreased WBC; foreign material, WBCs and bacteria on abdominal fluid, elevated lactate and decreased glucose on abdominal fluid	Medical unless perforation present
Target shaped soft tissue density on abdominal U/S, soft tissue density with gas dilation cranially on abdominal radiographs	Surgical (Immediate): medical management of primary cause
Colonic distension with hard feces on radiographs	Medical
Increased or decreased WBC, septic abdominal effusion	Surgical (Immediate)
Elevated T Bili, ALT, Alk Phos, and WBC hypoechoic hepatic parenchyma on ultrasound hepatomegaly	Medical after biopsy
Elevated T Bili, ALT, Alk Phos, and WBC hyperechoic foci in gallbladder or sludge on U/S, free gas in wall of gall bladder	Surgical (Immediate)
Abdominal effusion, bile pigment in effusion	Surgical (Immediate)
Elevated T Bili, Alk Phos, ALT	Surgical (Immediate)
Elevated or decreased WBC, elevated T Bili, Alk Phos and ALT, free gas in hepatic parenchyma on RADS, hypoechoic mass with hyperechoic material in hepatic parenchyma on U/S	Surgical (Immediate)
Heteroechoic liver with hyperechoic center on ultrasound	Surgical (Immediate)
Mixed echogenic mass on ultrasound, soft tissue mass density on radiographs, elevated alk phos, ALT, T Bili, hypoglycemia	Surgical (Immediate or delayed)

TABLE 1-24 Conditions That Can Cause Clinical Signs of Acute Abdominal Pain—cont'd

Diagnostic tests	Treatment
Elevated T Bili, Alk Phos, ALT, amylase and/or lipase, elevated or decreased WBC, hypocalcemia, focal loss of detail in right cranial quadrant on radiographs hypo- to hyperechoic pancreas with hyperechoic peri-pancreatic fat on ultrasound, abdominal and/or pleural effusion on radiographs and ultrasound	Medical in most cases unless abscess or phlegmon is present
Pancreatic soft tissue mass effect on radiographs and ultrasound, elevated amylase and lipase, hypoglycemia, elevated serum insulin	Surgical if mass identified, otherwise medical management of hypoglycemia
Splenomegaly on radiographs, hyperechoic spleen with no blood flow on ultrasound	Surgical (Immediate)
Soft tissue mass effect and loss of abdominal detail on radiographs, cavitated mass with abdominal effusion on U/S	Surgical (Immediate)
Hyperechoic spleen with no blood flow on abdominal U/S, abdominal effusion, thrombocytopenia	Surgical (Immediate)
Loss of abdominal detail on radiographs, peritoneal effusion on U/S, hemoabdomen on abdominocentesis	Medical unless refractory hypotension
Diagnosis based primarily on clinical signs	Medical
Fracture of the os penis on radiographs	Largely medical unless urethral tear
Diagnosis based primarily on clinical signs	Medical, although prepuce may need to be incised to allow replacement of penis into sheath
Prostatomegaly on radiographs and ultrasound hypoechoic prostate on U/S, pyuria and bacteriuria and U/A	Medical
Prostatomegaly on radiographs and ultrasound hypo- to hyperechoic prostate on U/S, bacteriuria and pyuria on U/A	Surgical (Delayed)
Prostatomegaly on radiographs and ultrasound, prostatic mineralization on radiographs and ultrasound	Medical/Surgical
Hypoechoic kidneys on U/S, pyuria on U/A, elevated WBC, azotemia	Medical
Pyuria, bacteriuria on U/A	Medical
Pyelectasia in abdominal U/S, azotemia	Surgical (Immediate)
Renomegaly on radiographs, azotemia	
Renal mass on U/S, renomegaly on radiographs	Surgical (Immediate)
Renal mass on U/S, azotemia, lack of renal blood flow on U/S	Surgical (Delayed)
Calculi in renal pelvis on radiographs and ultrasound, azotemia	Medical unless both kidneys affected
Ureteral calculi on radiographs and ultrasound, hydronephrosis, azotemia	Medical unless both kidneys affected
Ureteral calculi on radiographs and ultrasound, hydronephrosis, fluid or soft tissue density on U/S, azotemia	Surgical (Delayed until electrolyte stabilization)
Diagnosis largely based on physical examination findings	Medical unless cannot pass urethral catheter

Continued

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TABLE 1-24 Conditions That Can Cause Clinical Signs of Acute Abdominal Pain—cont'd

Diagnostic tests	Treatment
Azotemia, no peritoneal effusion, lack of urine output or outflow with ureteral catheterization, double contrast cystourethrogram indicated	Surgical (Delayed until electrolyte stabilization)
Transitional cellular casts on U/A, hematuria, mass effect or thickened irregular urethra on ultrasound or cystourethrogram	Surgical and medical management
Hypoechoic swollen testicle on testicular ultrasound	Surgical (Immediate)
Fluid or gas-filled tubular structure on abdominal ultrasound or abdominal radiographs	Surgical (Immediate)
Soft tissue tubular structure on radiographs, fluid-filled uterus on ultrasound, azotemia, isosthenuria, elevated T Bili, ALT and Alk Phos	Surgical (Immediate)
Pneumoperitoneum on radiographs, abdominal effusion, degenerative neutrophils and bacteria on abdominal fluid cytology	Surgical (Immediate)
Elevated WBC, increased density to bony endplates on radiographs	Medical
Hypocalcemia, markedly elevated CK	Medical
Diagnosis of exclusion	Medical
Decreased intervertebral disk space on radiographs, evidence of disk herniation and cord compression with myelogram or MRI	Surgical (Immediate)
Elevated protein and neutrophils on CSF analysis	Medical
Muscle biopsy	Medical
Mass effect and loss of abdominal detail on radiographs, mass and peritoneal effusion on ultrasound, anemia	Surgical (Immediate)
Degenerative neutrophils, plant material, bile pigment, or bacteria on abdominal fluid cytology; peritoneal effusion on U/S, loss of abdominal detail on radiographs, elevated or decreased WBC	Surgical (Immediate)
Elevated or decreased WBC, retroperitoneal mass effect on radiographs or ultrasound	Surgical (Immediate)

BOX 1-23 INDICATIONS TO PERFORM EXPLORATORY LAPAROTOMY

- Penetrating abdominal injury
- Presence of bacterial on abdominal fluid
- Presence of greater than 500 μ L of white blood cells in lavage fluid effluent, particularly if degenerative neutrophils are present
- Presence of food or plant material in lavage fluid
- Presence of creatinine, blood urea nitrogen, potassium, or lactate in abdominal fluid greater than that in peripheral blood
- Presence of glucose in abdominal fluid less than 50 mg/dL or less than that of peripheral blood
- Presence of bilirubin in lavage fluid
- Pneumoperitoneum on radiographs
- Continued evidence of peritoneal irritation

Diagnostic peritoneal lavage

In the event of a negative abdominocentesis, but peritoneal effusion or bile or gastrointestinal perforation are suspected, perform a diagnostic peritoneal lavage. Peritoneal dialysis kits are commercially available but are often expensive and impractical (see p. 6).

MANAGEMENT

Animals that have acute abdominal pain can be divided into three broad categories, depending on the primary cause of pain and the initial definitive treatment (Table 1-24). Some diseases warrant a nonsurgical, medical approach to case management. Other conditions require immediate surgery following rapid stabilization. Other conditions initially can be managed medically until the patient is hemodynamically more stable and then may or may not require surgical intervention at a later time. Specific management of each disease entity is listed under its own subheading.

Exploratory laparotomy/celiotomy

Box 1-23 lists specific indications for exploratory laparotomy. The best means to explore the abdominal cavity accurately and thoroughly is to open the abdomen on midline from the level of the xyphoid process caudally to the pubis for full exposure and then to evaluate all organs in every quadrant in a systematic manner. Address specific problems such as gastric or splenic torsion, enteroplication, and foreign body removal, and then copiously lavage the abdomen with warmed sterile saline solution. Suction the saline solution thoroughly from the peritoneal cavity so as to not impair macrophage function. In cases of septic peritonitis, the abdomen may be left open, or a drain may be placed for further suction and lavage. The routine use of antibiotics in irrigation solutions is contraindicated because the antibiotics can irritate the peritoneum and delay healing. When the abdominal cavity is left open, secure sterile laparotomy towels and water-impermeable dressings over the abdominal wound with umbilical tape, and then change these daily or as strike-through occurs. Open abdomen cases are often effusive and require meticulous evaluation and management of electrolyte imbalances and hypoalbuminemia. The abdomen can be closed and/or the abdominal drain removed when the volume of the effusion decreases, when bacteria are no longer present, and when the neutrophils become more healthy in appearance.

Additional Reading

- Bischoff MG: Radiographic techniques and interpretation of the acute abdomen, *Clin Tech Small Anim Pract* 18(1):7-19, 2003.
- Bonczynski JJ, Ludwig LL, Barton LJ, et al: Comparison of peritoneal fluid and peripheral blood pH, bicarbonate, glucose, and lactate as a diagnostic tool for septic peritonitis in dogs and cats, *Vet Surg* 32(2):161-166, 2003.
- Connally HE: Cytology and fluid analysis of the acute abdomen, *Clin Tech Small Anim Pract* 18(1):39-44, 2003.
- Cruz-Arambulo R, Wrigley R: Ultrasonography of the acute abdomen, *Clin Tech Small Anim Pract* 18(1):20-31, 2003.
- Herren V, Edwards L, Mazzaferro EM: Acute abdomen: diagnosis, *Compend Contin Educ Pract Vet* 26(5):350-363, 2004.
- Hofmeister EH: Anesthesia for the acute abdomen patient, *Clin Tech Small Anim Pract* 18(1):45-52, 2003.
- Mann FA: Acute abdomen: evaluation and emergency treatment. In Bonagura JD, editor: *Kirk's current veterinary therapy XIII*, Philadelphia, 2002, WB Saunders.
- Mazzaferro EM: Triage and approach to the acute abdomen, *Clin Tech Small Anim Pract* 18(1):1-6, 2003.
- Mueller MG, Ludwig LL, Barton LJ: Use of closed-suction drains to treat generalized peritonitis in dogs and cats: 40 cases (1997-1999), *J Am Vet Med Assoc* 219(6):789-794, 2001.
- Schmiedt C, Tobias KM, Otto CM: Evaluation of abdominal fluid: peripheral blood creatinine and potassium ratios for diagnosis of uroperitoneum in dogs, *J Vet Emerg Crit Care* 11(4):275-280, 2001.
- Walters JM: Abdominal paracentesis and diagnostic peritoneal lavage, *Clin Tech Small Anim Pract* 18(1):32-38, 2003.

1

ANAPHYLACTIC (ANAPHYLACTOID) SHOCK

Anaphylactic shock occurs as an immediate hypersensitivity reaction to a variety of inciting stimuli (Box 1-24). In animals, the most naturally occurring anaphylactic reaction results from wasp or bee stings. Most other reactions occur as a result of an abnormal sensitivity to items used in making medical diagnoses or treatment.

During an anaphylactic reaction, activation of C5a and the complement system results in vascular smooth muscle dilation and the release of a cascade of inflammatory mediators, including histamine, slow-reacting substance of anaphylaxis, serotonin, heparin, acetylcholine, and bradykinin.

Clinical signs associated with anaphylaxis differ between dogs and cats. In dogs, clinical signs may include restlessness, vomiting, diarrhea, hematochezia, circulatory collapse, coma, and death. In cats, clinical signs often are associated with respiratory system abnormalities. Clinical signs may include ptialism, pruritus, vomiting, incoordination, bronchoconstriction, pulmonary edema and hemorrhage, laryngeal edema, collapse, and death.

IMMEDIATE ACTION/TREATMENT

The most important steps to remember in any emergency is to follow the ABCs of Airway, Breathing, and Circulation. First, establish an airway through endotracheal intubation or emergency tracheostomy, if necessary. Concurrently, an assistant should establish vascular or intraosseous access to administer drugs and fluids (Box 1-25).

DIFFERENTIAL DIAGNOSIS

Differential diagnoses to consider for anaphylactic shock include the following:

- Any cause of vomiting, diarrhea
- Toxin
- Internal hemorrhage
- Congestive heart failure

BOX 1-24 INCITING ALLERGENS THAT CAN CAUSE ANAPHYLACTOID REACTIONS, ANGIONEUROTIC EDEMA, OR URTICARIA

- | | |
|--|-----------------|
| • Adrenocorticotrophic hormone | • Oxytocin |
| • Antihistamines/antitoxins (foreign serums) | • Penicillin |
| • Benzocaine | • Penicillinase |
| • Chloramphenicol | • Procaine |
| • Erythromycin | • Salicylates |
| • Food | • Streptomycin |
| • Heparin | • Tetracaine |
| • Hypersensitization and skin testing | • Tetracycline |
| • Insect stings | • Tranquilizers |
| • Insulin | • Vaccines |
| • Iodinated contrast media | • Vancomycin |
| • Lidocaine | • Vitamins |

BOX 1-25 IMMEDIATE TREATMENT OF ANAPHYLACTIC SHOCK

1. Administer epinephrine (0.01 mL/kg 1:1000 epinephrine IV or IO). If vascular access cannot be established, administer the epinephrine intramuscularly (0.2 to 0.5 mL/kg). Repeat epinephrine dose in 10 to 15 minutes if clinical signs are not resolving.
2. Start intravenous crystalloid fluids (Normosol-R, PlasmaLyte-M, lactated Ringer's solution) at one fourth of a calculated shock dose (90 mL/kg/hour in dogs, 44 mL/kg/hour in cats).
3. Administer a short-acting steroid (dexamethasone sodium phosphate [Dex-SP], 0.25 to 1.0 mg/kg IV).
4. Administer antihistamines. Administer diphenhydramine (0.5 mg/kg IM). Administer famotidine (0.5 to 1.0 mg/kg IV).

Lower airway disease
Upper airway obstruction

MANAGEMENT

The patient should be hospitalized until complete resolution of clinical signs. After initial stabilization and treatment, it is important to maintain vascular access and continue intravenous fluid therapy until the patient is no longer hypotensive, and vomiting and diarrhea have resolved. In cases of fulminant pulmonary hemorrhage and edema, administer supplemental oxygen until the patient is no longer hypoxemic or orthopneic on room air. Normalize and maintain blood pressure using positive inotropes (dobutamine, 3–10 µg/kg/minute CRI) or pressors (dopamine, 3 to 10 µg/kg/minute IV CRI; SEE SHOCK). If blood-tinged vomitus or diarrhea has been observed, administer antibiotics to decrease the risk of bacterial translocation and sepsis (cefoxitin, 22 mg/kg IV tid; metronidazole, 10 mg/kg IV tid). Also consider using gastroprotectant drugs (famotidine, 0.5 to 1.0 mg/kg IV; ranitidine, 0.5 to 2.0 mg/kg PO, IV, IM bid; sucralfate, 0.25 to 1.0 g PO tid; omeprazole, 0.7 to 1.0 mg/kg PO sid).

ANGIONEUROTIC EDEMA AND URTICARIA

A second and less serious form of allergic reaction is manifested as angioneurotic edema and urticaria. In most cases, clinical signs develop within 20 minutes of an inciting allergen. Although this type of reaction causes patient discomfort, it rarely poses a life-threatening problem. Most animals have mild to severe swelling of the maxilla and periorbital regions. The facial edema also may be accompanied by mild to severe generalized urticaria. Some animals may paw at their face, rub at their eyes, or have vomiting or diarrhea.

IMMEDIATE ACTION/TREATMENT

The treatment for angioneurotic edema involves suppressing the immune response by administration of short-acting glucocorticoid drugs and blocking the actions of histamine by the synergistic use of histamine₁ and histamine₂ receptor blockers (Box 1-26).

DIFFERENTIAL DIAGNOSIS

In some cases, the inciting cause is a known recent vaccination or insect sting. Many times, however, the inciting cause is not known and is likely an exposure to a stinging insect or arachnid. Differential diagnoses for acute facial swelling and/or urticaria include acetaminophen toxicity (cats), anterior caval syndrome, lymphadenitis, vasculitis, hypoalbuminemia, and contact dermatitis.

MANAGEMENT

Observe animals that have presented for angioneurotic edema for a minimum of 20 to 30 minutes after injection of the short-acting glucocorticoids and antihistamines. Monitor blood pressure to make sure that the patient does not have concurrent anaphylaxis and hypotension. After partial or complete resolution of clinical signs, the animal can be discharged to its owner for observation. In dogs, mild vomiting or diarrhea may occur within 1 to 2 days after this type of reaction. Wherever possible, exposure to the inciting allergen should be avoided.

BOX 1-26 IMMUNE RESPONSE SUPPRESSION AGENTS FOR ANGIONEUROTIC EDEMA

- Administer short-acting glucocorticoid:
- Dexamethasone sodium phosphate (Dex-SP), 0.25 to 1.0 mg/kg IV, SQ, IM
- Administer antihistamines:
- Diphenhydramine, 0.5 to 1.0 mg/kg IM, SQ
- Famotidine, 0.5 to 1.0 mg/kg IV, SQ, IM

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Additional Reading

- Cohen R: Systemic anaphylaxis. In Bonagura J, editor: *Current veterinary therapy XII. Small animal practice*, Philadelphia, 1995, WB Saunders.
- Friberg CA, Lewis DT: Insect hypersensitivity in small animals, *Compend Contin Educ Pract Vet* 20(10):1121-1131, 1998.
- Meyer EK: Vaccine-associated adverse events, *Vet Clin North Am Small Anim Pract* 31(3): 493-514, 2001.
- Noble SJ, Armstrong PJ: Bee sting envenomation resulting in secondary immune-mediated hemolytic anemia in two dogs, *J Am Vet Med Assoc* 214(7):1026-1027, 1999.
- Plunkett SJ: Anaphylaxis to ophthalmic medication in a cat, *J Vet Emerg Crit Care* 10(3):169-171, 2000.
- Waddell LS, Drobatz KJ: Massive envenomation by *Vespula* spp in two dogs, *J Vet Emerg Crit Care* 9(2):67-71, 1999.

ANESTHETIC COMPLICATIONS AND EMERGENCIES

Complications observed while a patient is under anesthesia can be divided into two broad categories: (1) those related to equipment malfunction or human error and (2) the patient's physiologic response to the cardiorespiratory effects of the anesthetic drugs. Careful observation of the patient and familiarity with anesthetic equipment, drug protocols, and monitoring equipment is necessary for the safest anesthesia to occur. Despite this, however, anesthetic-related complications are frequent and need to be recognized and treated appropriately.

THE RESPIRATORY SYSTEM

Many anesthetic drugs have a dose-dependent depressive effect on the respiratory system and cause a decrease in respiratory rate and tidal volume, leading to hypoventilation. Respiratory rate alone is not a reliable indicator of the patient's oxygenation and ventilatory status. The respiratory tidal volume can be measured with a Wright's respirometer. Perform pulse oximetry and capnography as noninvasive measures of the patient's oxygenation and ventilation.

Ventilation can be impaired as a result of anesthetic drugs, patient position, pneumothorax, pleural effusion (chylothorax, hemothorax, pyothorax), equipment malfunction, rebreathing of carbon dioxide, thoracic wall injury, or alveolar fluid (pulmonary edema, hemorrhage, or pneumonia). Problems such as a diaphragmatic hernia, GDV, or gravid uterus can impede diaphragmatic excursions once the patient is placed on its back and can lead to impaired ventilation. The work of breathing also may be increased because of increased resistance of the anesthesia circuit and increased dead space ventilation. This is particularly important in small toy breeds.

Clinical signs of inadequate ventilation and respiratory complications include abnormal respiratory pattern, sudden changes in heart rate, cardiac dysrhythmias, cyanosis, and cardiopulmonary arrest. End-tidal carbon dioxide, or capnography, gives a graphic display of adequacy of ventilation. Rapid decreases in end-tidal carbon dioxide can be caused by disconnection or obstruction of the patient's endotracheal tube or poor perfusion, namely, cardiopulmonary arrest (see Capnometry [End-Tidal Carbon Dioxide Monitoring]).

Postoperatively, hypoventilation can occur because of the residual effects of the anesthetic drugs, hypothermia, overventilation during intraoperative support, surgical techniques that compromise ventilation (thoracotomy, cervical disk surgery, atlantooccipital stabilization), postoperative bandaging of the abdomen or thorax, ventilatory muscle fatigue, or injury to the CNS.

CARDIOVASCULAR SYSTEM

Cardiac output is a function of heart rate and stroke volume. Factors that influence stroke volume include vascular and cardiac preload, cardiac afterload, and cardiac contractility. The patient's cardiac output can be affected adversely by the negative inotropic and chronotropic and vasodilatory effects of anesthetic drugs, all leading to hypotension.

Bradycardia, tachycardia, cardiac dysrhythmias, and vascular dilation can lead to hypotension and inadequate organ perfusion. Table 1-25 lists the normal heart rate and blood pressure in dogs and cats.

Bradycardia

Bradycardia is defined as a heart rate below normal values. Many anesthetic drugs can cause bradycardia. Causes of bradycardia include the use of narcotics or α_2 -agonist drugs, deep plane of anesthesia, increased vagal tone, hypothermia, and hypoxia. Table 1-26 lists the causes of bradycardia and the necessary immediate action or treatment.

Tachycardia

Tachycardia is defined as a heart rate above normal values. Common causes of tachycardia include vasodilation, drugs, inadequate anesthetic depth and perceived pain, hypercapnia, hypoxemia, hypotension, shock, or hyperthermia. Table 1-27 lists the causes and immediate action or treatment for tachycardia.

Hypotension

Hypotension is defined as physiologically low blood pressure (mean arterial pressure less than 65 mm Hg). A mean arterial blood pressure less than 60 mm Hg can result in inadequate tissue perfusion and oxygen delivery. The coronary arteries are perfused during diastole. Inadequate diastolic blood pressure, less than 40 mm Hg, can cause decreased coronary artery perfusion and myocardial hypoxemia that can predispose the heart to dysrhythmias. Causes of perianesthetic hypotension include peripheral vasodilation by anesthetic drugs, bradycardia or tachyarrhythmias, hypothermia, inadequate cardiac preload from vasodilation or hemorrhage, decreased venous return from patient position or surgical manipulation of viscera, and decreased cardiac contractility. Table 1-28 lists possible causes of hypotension and immediate actions to take.

TABLE 1 - 25 Normal Parameters for Heart Rate and Blood Pressure in Dogs and Cats

Species	Normal heart rate (beats/minute)	Normal blood pressure (mm Hg)		
		Systolic	Diastolic	Mean
Dogs (large)	60-100	100-160	60-90	80-120
Dogs (medium)	80-120			
Dogs (small)	90-140			
Cats	140-200	100-160	60-90	80-120

TABLE 1 - 26 Causes and Treatment of Bradycardia

Cause	Immediate action
Anesthetic drug:	
Opioid	Reverse effects with naloxone.
α_2 -Agonist	Reverse effects with yohimbine or atipamezole.
Deep anesthesia	Decrease vaporizer setting.
Increased vagal tone	Administer a parasympatholytic (atropine or glycopyrrolate).
Hypothermia	Provide ambient rewarming.
Hypoxia	Provide supplemental oxygen.

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TABLE 1-27 Causes and Treatment of Tachycardia

Cause	Immediate action
Vagolytic drugs	
Atropine	Allow time for the drug to wear off.
Glycopyrrolate	Allow time for the drug to wear off.
Sympathomimetic drugs	
Epinephrine	Allow time for the drug to wear off; administer a β -blocker; turn off infusion.
Isoproterenol	Administer a β -blocker.
Dopamine	Turn off infusion; administer a β -blocker.
Ketamine	Allow time for drug to wear off.
Inadequate anesthetic depth	Increase anesthetic depth.
Hypercapnia	Increase ventilation (assisted ventilation).
Hypoxemia	Increase gas flow and oxygenation.
Hypotension	Decrease anesthetic depth; administer an intravenous crystalloid or colloid bolus, positive inotrope drug, positive chronotrope drug, or pressor.
Hyperthermia	Apply ambient or active cooling measures; administer dantrolene sodium if malignant hyperthermia is suspected.

TABLE 1-28 Causes and Treatment of Perianesthetic Hypotension

Cause	Immediate action
Hypothermia	Provide ambient rewarming.
Hypocalcemia*	Administer calcium chloride (10 mg/kg IV) or calcium gluconate (23 mg/kg).
Increased anesthetic	Decrease vaporizer setting/anesthetic depth. Reverse with opioids or α_2 -agonists.
Vasodilation	Administer an intravenous crystalloid bolus (10 mL/kg). Administer an intravenous colloid bolus (5 mL/kg). Administer a pressor (epinephrine, phenylephrine).
Negative inotropy	Decrease anesthetic depth. Administer ephedrine (0.1-0.25 mg/kg IV). Administer dobutamine (2-20 μ g/kg IV CRI). Administer dopamine (2-10 μ g/kg/minute). Administer norepinephrine (0.05-0.4 μ g/kg/minute IV CRI). Administer epinephrine (0.05-0.4 μ g/kg/minute IV CRI).
Bradycardia	Administer atropine (0.01-0.04 mg/kg IV or SQ). Administer glycopyrrolate (0.005-0.02 mg/kg IV, SQ).

*Hypocalcemia caused by chelation from EDTA with multiple blood product transfusion (cats are particularly susceptible).

Cardiac dysrhythmias

Electrocardiogram monitoring is useful for the early detection of cardiac dysrhythmias during the perianesthetic period. Clinical signs of cardiac dysrhythmias include irregular pulse rate or pressure, abnormal or irregular heart sounds, pallor, cyanosis, hypotension, and an abnormal ECG tracing. Remember that the single best method of detecting cardiac

dysrhythmias is with your fingertips (palpate a pulse or apex heartbeat) and ears (auscultate the heart). Confirm the dysrhythmia by auscultating the heart rate and rhythm, identify the P waves and the QRS complexes, and evaluate the relationship between the P waves and QRS complexes. Is there a P wave for every QRS, and a QRS for every P wave? During anesthesia, fluid, acid-base, and electrolyte imbalances can predispose the patient to dysrhythmias. Sympathetic and parasympathetic stimulation, including the time of intubation, can predispose the patient to dysrhythmias. If the patient's plane of anesthesia is too light, perception of pain can cause catecholamine release, sensitizing the myocardium to ectopic beats. Atrioventricular blockade can be induced with the administration of α_2 -agonist medications, including xylazine and medetomidine. Thiobarbiturates (thiopental) can induce ventricular ectopy and bigeminy. Although these dysrhythmias may not be harmful in the awake patient, anesthetized patients are at a particular risk of dysrhythmia-induced hypotension. Carefully monitor and treat all dysrhythmias (see Cardiac Dysrhythmias). Box 1-27 lists steps to take to prevent perianesthetic dysrhythmias.

DEPTH EVALUATION AND HUMAN ERROR

Awakening during anesthesia can occur and can be caused by equipment failure and simply, although no one likes to admit it, human error. Table 1-29 lists causes of arousal during anesthesia and appropriate immediate actions.

BOX 1-27 STEPS TO PREVENT PERIANESTHETIC DYSRHYTHMIAS

- Stabilize acid-base and electrolyte balance before anesthetic induction, whenever possible.
- Rehydrate patient before anesthetic induction.
- Select anesthetic agents appropriate for the particular patient.
- Be aware of the effects of the drugs on the myocardium.
- Ensure adequate anesthetic depth and oxygenation before anesthetic induction.
- Ensure ventilatory support during anesthesia.
- Monitor heart rate, rhythm, blood pressure, pulse oximetry, and capnometry during anesthesia.
- Ensure adequate anesthetic depth before surgical stimulation.
- Avoid surgical manipulation to the heart or great vessels, whenever possible.
- Avoid changes in perianesthetic depth.
- Avoid hypothermia.

TABLE 1-29 Causes and Treatment of Arousal during Anesthesia

Cause	Immediate action
Postinduction hypoventilation	Increase ventilatory rate and volume.
Too low an inspired gas flow	Increase anesthetic gas flow.
Fresh gas flow too low	Increase fresh gas flow.
Equipment malfunction in machine or vaporizer	Change anesthetic machines.
Esophageal intubation	Reintubate into trachea, and check placement with capnometry or laryngoscope.
Undersized endotracheal tube with leaks	Replace with appropriate sized endotracheal tube.
Inadequate endotracheal cuff inflation	Inflate endotracheal cuff appropriately to decrease leaks.
Surgical stimulus	Increase depth of anesthesia.
Conditions that mimic anesthetic arousal (e.g., malignant hyperthermia)	Awaken patient, and administer dantrolene sodium.

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POSTANESTHETIC COMPLICATIONS

Delayed recovery can be caused by a number of factors, including excessive anesthetic depth, hypothermia, residual action of narcotics or tranquilizers, delayed metabolism of anesthetic drugs, hypoglycemia, hypocalcemia, hemorrhage, and breed or animal predisposition. Careful monitoring of the patient's blood pressure, acid-base and electrolyte status, anesthetic depth, PCV, and vascular volume intraoperatively and taking care with supportive measures to prevent abnormalities can hasten anesthetic recovery and avoid postoperative complications.

Additional Reading

Gaynor JS, Muir WW: *Handbook of veterinary pain management*, St Louis, 2003, Mosby.
Mazzaferro EM, Wagner AE: Hypotension during anesthesia in dogs and cats: recognition, causes, and treatment, *Compend Contin Educ Pract Vet* 23(8):728-737, 2001.
Melzack R, Wall PD: *Handbook of pain management: a clinical companion to Wall and Melzack's textbook of pain*, Edinburgh, 2003, Churchill Livingstone.
Muir WW, Hubbell JAE, Skarda RT, et al: *Handbook of veterinary anesthesia*, ed 3, St Louis, 2000, Mosby.
Thurmon JC, Tranquilli WJ, Benson GJ: *Lumb and Jones' veterinary anesthesia*, ed 3, Philadelphia, 1996, Lippincott Williams & Wilkins.

BLEEDING DISORDERS

The presentation of a patient with a bleeding disorder often is a diagnostic challenge for the veterinary practitioner (Boxes 1-28 and 1-29). In general, abnormal bleeding can be caused by five major categories: (1) vascular trauma, (2) defective production of hemostatic factors, (3) dilution of hemostatic factors, (4) use or toxicity of systemic anticoagulants, and (5) DIC. A clotting disorder should be suspected in any patient with a history of

BOX 1-28 CAUSES OF DEFECTIVE PRIMARY HEMOSTASIS	
THROMBOCYTOPENIA	Systemic illness
Impaired or defective thrombopoiesis	Uremia
Immune-mediated destruction	Pancreatitis
Myelophthisis	Ehrlichiosis
Drug-induced	Dysproteinemias
Decreased platelet life span in circulation	Myeloproliferative and myelodysplastic disorders
Antibody-mediated platelet destruction	Disseminated intravascular coagulation
Consumption in disseminated intravascular coagulopathy	Antiplatelet drugs
	Aspirin
THROMBOCYTOPATHIA	
Congenital illness	
Von Willebrand's disease	
Other hereditary thrombocytopathies	

BOX 1-29 CAUSES OF DEFECTIVE SECONDARY HEMOSTASIS	
Clotting factor deficiency	Circulating inhibitors of coagulation
Decreased production	Heparin
Hereditary causes	Fibrin degradation products
Chronic hepatic insufficiency/failure	
Vitamin K antagonist rodenticides	
Increased consumption	
Disseminated intravascular coagulation	
Hemangiosarcoma	

development of spontaneous deep hematomas, unusually prolonged bleeding after traumatic injury, bleeding at multiple sites throughout the body involving multiple organ systems, delayed onset of severe hemorrhage after bleeding, and an inability on the practitioner's part to find an organic cause of bleeding. The signalment, history, clinical signs, and results of coagulation often can aid in making a rapid diagnosis of the primary cause of the disorder and in the selection of appropriate case management. When taking a history, ask the following important questions:

- What is the nature of the bleeding?
- What sites are affected?
- How long has the bleeding been going on?
- Has your animal had any previous or similar episodes?
- Is there any possibility of any toxin exposure?
- If so, when and how much did your animal consume?
- Is there any possibility of trauma?
- Does your animal run loose outdoors unattended?
- Have you ever traveled, and if so, where?
- Has your animal been on any medications recently or currently?
- Has your animal been vaccinated recently?
- Have any known relatives of your animal had any bleeding disorders?
- Are there any other abnormal signs that you have seen?

Abnormalities found on physical examination may aid in determining whether the hemorrhage is localized or generalized (i.e., bleeding from a venipuncture site versus bleeding diathesis). Note whether the clinical signs are associated with a platelet problem and superficial hemorrhage or whether deep bleeding can be associated with abnormalities of the coagulation cascade. Also, make an attempt to identify any concurrent illness that can predispose the patient to a bleeding disorder (i.e., pancreatitis, snakebite, sepsis, immune-mediated hemolytic anemia, or severe trauma and crush or burn injury).

Abnormalities associated with coagulopathies include petechiae and ecchymoses, epistaxis, gingival bleeding, hematuria, hemarthrosis, melena, and hemorrhagic cavity (pleural and peritoneal or retroperitoneal) effusions.

SPECIFIC COAGULOPATHIES

Disseminated intravascular coagulation (DIC)

Disseminated intravascular coagulation is a complex syndrome that results from the inappropriate activation of the clotting cascade, leading to disruption of the normal balance between thrombosis and fibrinolysis. The formation of diffuse microthrombi with concurrent consumption of platelets and activated clotting factors leads to end-organ thrombosis with various degrees of clinical hemorrhage. In animals, DIC always results from some other pathologic process, including various forms of neoplasia, crush and heat-induced injury, sepsis, inflammation, and immune-mediated disorders (Box 1-30). The pathophysiologic mechanisms involved in DIC include vascular endothelial damage, activation and consumption of platelets, release of tissue procoagulants, and consumption of endogenous anticoagulants.

Diagnosis of disseminated intravascular coagulation

Because DIC always results from some other disease process, diagnosis of DIC is based on a number of criteria when evaluating various coagulation tests, peripheral blood smears, platelet count, and end products of thrombosis and fibrinolysis.

There is no one definitive criterion for the diagnosis of DIC (Box 1-31). Thrombocytopenia occurs as platelets are consumed during thrombosis. It is important to remember that trends in decline in platelet numbers are just as important as thrombocytopenia when making the diagnosis. In some cases the platelet count still may be within the normal reference range but has significantly decreased in the last 24 hours. Early in DIC the procoagulant cascade dominates, with hypercoagulability. Activated clotting time, APTT, and PT may be rapid and shorter than normal. In most cases, we do not recognize the

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BOX 1-30 DISORDERS ASSOCIATED WITH DISSEMINATED INTRAVASCULAR COAGULATION IN THE DOG

Neoplasia	Heartworm disease
Hemangiosarcoma	Immune-mediated disease
Lymphoma	Immune-mediated hemolytic anemia
Inflammation	Trauma
Pancreatitis	Crush injury
Heat-induced injury	Burn injury
Gastric dilatation-volvulus	Snake envenomation
Mesenteric torsion	
Sepsis	
Gram-negative and gram-positive sepsis	
Septic peritonitis	
Gangrenous mastitis	
Pyothorax	

BOX 1-31 LABORATORY FINDINGS ASSOCIATED WITH A DIAGNOSIS OF DISSEMINATED INTRAVASCULAR COAGULATION*

- Red blood cell fragments
- Thrombocytopenia
- Rapid or prolonged activated partial thromboplastin time
- Rapid or prolonged prothrombin time
- Rapid or prolonged activated clotting time
- Hypofibrinogenemia
- Positive fibrin degradation products without concurrent hepatic disease
- Decrease in antithrombin concentration
- Positive D-dimer test

*More than one of the above criteria should be present to aid in the diagnosis of disseminated intravascular coagulation.

hypercoagulable state in our critically ill patients. Later in DIC, as platelets and activated clotting factors become consumed, the ACT, APTT, and PT become prolonged. Antithrombin, a natural anticoagulant, also becomes consumed, and antithrombin levels decline. Antithrombin levels can be measured at commercial laboratories and in some large veterinary institutions. The end products of thrombosis and subsequent fibrinolysis also can be measured. Fibrinogen levels may decline, although this test is not sensitive or specific for DIC. Fibrin degradation (split) products also become elevated. Fibrin degradation products are normally cleared by the liver, and these also become elevated in cases of hepatic failure because of lack of clearance. More recently, cageside D-dimer tests have become available to measure the breakdown product of cross-linked fibrin as a more sensitive and specific monitor of DIC.

Management of disseminated intravascular coagulation

Management of DIC first involves treating the primary underlying cause. By the time DIC becomes evident, rapid and aggressive treatment is necessary. If you are suspicious of DIC in any patient with a disease known to incite DIC, then ideally, you should begin treatment BEFORE the hemostatic abnormalities start to occur for the best possible prognosis. Treatment involves replacement of clotting factors and antithrombin and prevention of further clot formation. To replenish clotting factors and antithrombin, administer fresh whole blood or fresh frozen plasma. Heparin requires antithrombin as a cofactor to inactivate thrombin and other activated coagulation factors. Administer heparin (50 to 100 units/kg SQ

q6-8h of unfractionated heparin; or fractionated enoxaparin [Lovenox], 1 mg/kg SQ bid). Aspirin (5 mg/kg PO bid in dogs; every third day in cats) also can be administered to prevent platelet adhesion. Management of DIC also involves the rule of twenty monitoring and case management to maintain end-organ perfusion and oxygen delivery (see the Rule of 20).

CONGENITAL DEFECTS OF HEMOSTASIS

Factor VIII deficiency (hemophilia A)

Hemophilia A is a sex-linked recessive trait that is carried by females and manifested in males. Female hemophiliacs can occur when a hemophiliac male is bred with a carrier female. Hemophilia A has been reported in cats and a number of dog breeds, including Miniature Schnauzer, Saint Bernard, Miniature Poodle, Shetland Sheepdog, English and Irish Setters, Labrador Retriever, German shepherd, Collie, Weimaraner, Greyhound, Chihuahua, English bulldog, Samoyed, and Vizsla. Mild to moderate internal or external bleeding can occur. Clinical signs of umbilical cord bleeding can become apparent in some animals shortly after weaning. Gingival hemorrhage, hemarthrosis, gastrointestinal hemorrhage, and hematomas may occur. Clotting profiles in animals with factor VIII deficiency include prolonged APTT and ACT. The PT and buccal mucosa bleeding time are normal. Affected animals have low factor VIII activity but normal to high levels of factor VIII-related antigen. Carrier females can be detected by low (30% to 60% of normal) factor VIII activity and normal to elevated levels of factor VII-related antigen.

Von Willebrand's disease

Von Willebrand's disease is a deficiency or defect in von Willebrand's protein. A number of variants of the disease have been described: Von Willebrand's disease type I is associated with a defect in factor VIII/protein concentration, and von Willebrand's disease type II is associated with a defect in VIII:vWF. Type I von Willebrand's disease is most common in veterinary medicine. Von Willebrand's disease has been identified in more than 29 breeds of dogs, with an incidence that varies from 10% to 60% depending on the breed of origin. Affected breeds include Doberman Pinchers, German Shepherd Dogs, Scottish Terriers and standard Manchester Terriers, Golden Retrievers, Chesapeake Bay Retrievers, Miniature Schnauzers, and Pembroke Welsh Corgis. Two forms of genetic expression occur: (1) autosomal recessive disease in which homozygous von Willebrand's disease individuals have a bleeding disorder, whereas heterozygous individuals carry the trait but are clinically normal. The second variant of genetic expression involves an autosomal dominant disease with incomplete expression such that heterozygous individuals are affected carriers and homozygous individuals are severely affected. Von Willebrand's disease has high morbidity, but fortunately a low mortality. Dogs with 30% or less than normal vWF tend to hemorrhage. Platelet counts are normal, but bleeding times can be prolonged. The APTT can be slightly prolonged when factor VIII is less than 50% of normal. Routine screening tests are nondiagnostic for this disease, although in a predisposed breed with a normal platelet count, a prolonged buccal mucosa bleeding time strongly supports a diagnosis of von Willebrand's disease. Documentation of clinical bleeding with low or undetectable levels of factor VIII antigen or platelet-related activities of vWF support a diagnosis of von Willebrand's disease. Recessive animals have zero vWF:antigen (a subunit of factor III); heterozygotes have 15% to 60% of normal. In the incompletely dominant form, levels of vWF antigen are reduced (less than 7% to 60%). Clinical signs in affected animals include epistaxis, hematuria, diarrhea with melena, penile bleeding, lameness, hemarthrosis, hematoma formation, and excessive bleeding with routine procedures such as nail trimming, ear cropping, tail docking, surgical procedures (spay, neuter), and lacerations. Estrous and postpartum bleeding may be prolonged. A DNA test to detect carriers of the vWF gene is available through VetGen (Ann Arbor, Michigan) and Michigan State University. Patients with von Willebrand's disease should avoid drugs known to affect platelet function adversely (sulfonamide, ampicillin, chloramphenicol, antihistamines, theophylline, phenothiazine tranquilizers, heparin, and estrogen).

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Factor IX (Christmas factor) deficiency (hemophilia B)

Hemophilia B is an X-linked recessive trait that occurs with less frequency than hemophilia A. The disease has been reported in Scottish Terriers, Shetland and Old English Sheepdogs, Saint Bernards, Cocker Spaniels, Alaskan Malamutes, Labrador Retrievers, Bichon Frises, Airedale Terriers, and British Shorthair cats. Carrier females have low (40% to 60% of normal) factor IX activity. Clinical signs are more severe than for hemophilia A.

Factor VII deficiency

Congenital deficiencies of factor VII have been reported as an autosomal, incompletely dominant characteristic in Beagles. Heterozygotes have 50% factor VII deficiency. Bleeding tends to be mild. The PT is prolonged in affected individuals.

Factor X deficiency

Factor X deficiency has been documented in Cocker Spaniels and resembles fading-puppy syndrome in newborn dogs. Internal or umbilical bleeding can occur, and affected dogs typically die. Bleeding may be mild in adult dogs. In severe cases, factor X levels are reduced to 20% of normal; in mild cases, factor X levels are 20% to 70% of normal.

Factor XII (Hageman factor) deficiency

Factor XII deficiency has been documented as an inherited autosomal recessive trait in domestic cats. Heterozygotes can be detected because they have a partial deficiency (50% of normal) of factor XII. Homozygote cats have less than 2% factor XII activity. Deficiency of Hageman factor usually does not result in bleeding or other disorders.

Factor XI deficiency

Factor XI deficiency is an autosomal disease that has been documented in Kerry Blue Terriers, Great Pyrenees, and English Springer Spaniels. In affected individuals, protracted bleeding may be observed. Homozygotes have low factor XI activity (< 20% of normal), and heterozygotes have 40% to 60% of normal.

Management of congenital defects of hemostasis

The management of congenital defects of hemostasis typically involves replenishing the clotting factor that is present. Usually, this can be accomplished in the form of fresh frozen plasma transfusion (20 mL/kg). If anemia is present because of severe hemorrhage, fresh whole blood or packed RBCs also can be administered. Recent research has investigated the use of recombinant gene therapy in the treatment of specific factor deficiencies in dogs; however, the therapy is not yet available for use in clinical practice.

In cases of von Willebrand's disease, administration of fresh frozen plasma (10 to 20 mL/kg) or cryoprecipitate (1 unit/10 kg body mass) provides vWF, factor VIII, and fibrinogen. Doses can be repeated until hemorrhage ceases. 1-Desamino-8-D-arginine vasopressin (DDAVP) also can be administered (1 µg/kg SC or IV diluted in 0.9% saline given over 10 to 20 minutes) to the donor and patient to increase the release of stored vWF from endothelial cells. A fresh whole blood transfusion can be obtained from the donor and immediately administered to the patient, or spun down and the fresh plasma administered if RBCs are not needed. Administer a dose of DDAVP to any affected dog before initiating any elective surgical procedures. A supply of fresh frozen plasma and RBCs should be on hand, should uncontrolled hemorrhage occur.

ACQUIRED DISORDERS OF HEMOSTASIS

Platelets are essential to normal blood coagulation. After a vessel is damaged, release of vasoactive amines causes vasoconstriction and sluggish flow of blood in an attempt to squelch hemorrhage. Platelets become activated by platelet activating factor, and attach to the damaged vascular endothelium. Normal platelet adhesion depends on mediators such as calcium, fibrinogen, vWF:antigen, and a portion of factor VIII. After adhesion, the platelets undergo primary aggregation and release a variety of chemical mediators

including adenosine diphosphate, prostaglandins, serotonin, epinephrine, thromboplastin, and thromboxane A that promote secondary aggregation and contraction. Platelet abnormalities can include decreased platelet production (thrombocytopenia), decreased platelet function (thrombocytopathia), increased platelet destruction, increased platelet consumption, and platelet sequestration.

Thrombocytopathia

Thrombocytopathia refers to platelet function abnormalities. Alterations in platelet function can affect platelet adhesion, aggregation, or release of vasoactive substances that help form a stable clot (Box 1-32). In von Willebrand's disease there is a deficiency in vWF:antigen that results in altered platelet adhesion. Vascular purpuras are reported and have been seen in collagen abnormalities such as Ehlers-Danlos syndrome, which can be inherited as an autosomal dominant trait with complete penetrance and has been recognized in German Shepherd Dogs, Dachshunds, Saint Bernards, and Labrador Retrievers.

Thrombasthenic thrombopathy is a hereditary autosomal dominant abnormality that has been described in Otterhounds, Foxhounds and Scottish Terriers. In this condition, platelets do not aggregate normally in response to adenosine diphosphate and thrombin stimulation.

Evaluation of platelet function is based on a total platelet count, buccal mucosa bleeding time, and thromboelastography. Platelet function defects (thrombocytopenia and thrombocytopathia) can affect both sexes. Clinical signs can resemble von Willebrand's disease. In most cases, buccal mucosa bleeding time will be prolonged, but platelet count and clotting tests will be normal.

BOX 1-32 CAUSES OF THROMBOCYTOPATHIA

DRUGS

Aspirin or other nonsteroidal antiinflammatory drugs
Heparin
Phenothiazine tranquilizers
Cephalosporin

SYSTEMIC DISORDERS

Uremia
Liver disease

HEMATOLOGIC DISORDERS

Antiplatelet antibody production
Myeloproliferative disorders
Dysproteinemia
von Willebrand's disease defects

INHERITED

Thrombasthenic thrombopathy of Otterhounds
Glanzmann's thrombasthenia of Great Pyrenees
Thrombopathy of Bassett Hound and American Eskimo Dog (Spitz)
Cyclic hematopoiesis of the gray Collie
Platelet storage pool disease of American Cocker Spaniel

Thrombocytopenia

Platelet count can be decreased because of problems with production, increased consumption, sequestration, or destruction. Causes of accelerated platelet destruction are typically immune-mediated autoantibodies, drug antibodies, infection, and isoimmune destruction. Consumption and sequestration usually are caused by DIC, vasculitis, microangiopathic hemolytic anemia, severe vascular injury, hemolytic uremic syndrome, and gram-negative septicemia. Primary thrombocytopenia with no known cause has been called *idiopathic thrombocytic purpura*. In approximately 80% of the cases, thrombocytopenia is associated with immune-mediated destruction caused by immune-mediated hemolytic anemia, systemic lupus erythematosus, rheumatoid arthritis, DIC, and diseases that affect the bone marrow. In systemic lupus erythematosus, 20% to 30% of the affected dogs have concurrent

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idiopathic thrombocytic purpura. When immune-mediated hemolytic anemia and idiopathic thrombocytic purpura are present in the same patient, the disease is called *Evans syndrome*. PF-3 is a non-complement-fixing antibody that is produced in the spleen and affects peripheral and bone marrow platelets and megakaryocytes. Antibodies directed against platelets are usually of the IgG subtype in animals. Antiplatelet antibodies can be measured by a PF-3 release test. Platelet counts with immune-mediated destruction typically are less than 50,000 platelets/ μ L. Infectious causes of thrombocytopenia include *Ehrlichia canis*, *Anaplasma phagocytophilum* (formerly, *Ehrlichia equi*), and *Rickettsia rickettsii* (Rocky Mountain spotted fever). Primary immune-mediated thrombocytopenia has an unknown cause and most frequently is seen in middle- to older-aged female dogs. Breed predispositions include Cocker Spaniels, German Shepherd Dogs, Poodles (toy, miniature, standard), and Old English Sheepdogs.

Thrombocytopenia usually is manifested as petechiae, ecchymoses of skin and mucous membranes, hyphema, gingival and conjunctival bleeding, hematuria, melena, and epistaxis. To make a diagnosis of idiopathic thrombocytic purpura, measure the severity of thrombocytopenia ($< 50,000$ platelets/ μ L), analyze the peripheral blood smear for evidence of platelet fragmentation or microthrombocytosis, normal to increased numbers of megakaryocytes in the bone marrow, detection of antiplatelet antibody, increased platelet counts after starting glucocorticoid therapy, and elimination of other causes of thrombocytopenia. If tick-borne illnesses are suspected, antibody titers for *E. canis*, *A. phagocytophilum* (formerly *E. equi*), and *R. rickettsii* should be performed.

Treatment of immune-mediated thrombocytopenia involves suppression of the immune system to stop the immune-mediated destruction and to stimulate platelet release from the bone marrow. Traditionally, the gold standard to suppress the immune system is to use glucocorticoids (prednisone or prednisolone, 2 to 4 mg/kg PO bid divided, OR dexamethasone, 0.1 to 0.3 mg/kg IV or PO q12h). More recently human serum immunoglobulin (IgG) also has been used (0.2 to 0.5 g/kg IV in saline over 8 hours; pretreat with 1 mg/kg diphenhydramine 15 minutes before starting infusion). Vincristine (0.5 mg/m² IV once) can stimulate the release of platelets from the bone marrow if megakaryocytic precursors are present; however, the platelets released may be immature and potentially nonfunctional. Treatment with fresh whole blood or packed RBCs is appropriate if anemia is present; however, unless specific platelet-rich plasma has been purchased from a blood bank, fresh whole blood contains relatively few platelets, which are short-lived (2 hours) and will not effectively raise the platelet count at all. Finally, long-term therapy is usually in the form of azathioprine (2 mg/kg PO once daily, tapered to 1 mg/kg daily to every other day after 1 week) and cyclosporine (10 to 25 mg/kg PO divided). If a tick-borne illness is suspected, administer doxycycline (5 to 10 mg/kg PO bid) for 4 weeks or if titers come back negative.

Thrombocytopenia also can occur in the cat. Causes for thrombocytopenia in cats include infections (29%), neoplasia (20%), cardiac disease (7%), primary immune-mediated disease (2%), and unknown causes (20%). In one study of cats with feline leukemia and myeloproliferative disease, 44% of cases had thrombocytopenia.

Vitamin K antagonist rodenticide intoxication

Warfarin and coumarin derivatives are the major class of rodenticides used in the United States. Vitamin K antagonist rodenticides inhibit the epoxidase reaction and deplete active vitamin K, causing a depletion of vitamin K-dependent coagulation factors (II, VII, IX, X) within 24 hours to 1 week of ingestion, depending on the ingested dose. Affected animals can spontaneously hemorrhage anywhere in the body. Clinical signs can include hemoptysis, respiratory difficulty, cough, gingival bleeding, epistaxis, hematuria, hyphema, conjunctival bleeding, petechiae and ecchymoses, cavity hemorrhage (pleural, peritoneal, retroperitoneal) with acute weakness, lethargy or collapse, hemarthrosis with lameness, deep muscle bleeds, and intracranial or spinal cord hemorrhage. Diagnosis of vitamin K antagonism includes prolonged PT. A PIVKA (protein induced by vitamin K absence or antagonism) test also can be performed, if possible.

TABLE 1-30 Clinical Interpretation of Laboratory Test Results for Coagulation Profiles

Disorder	BMBT	ACT	PT	APTT	Platelets	Fibrinogen	FDPs	D-dimers
Thrombocytopenia	↑	N	N	N	↓	N	N	N
Thrombocytopathia	↑	N	N	N	N	N	N	N
Von Willebrand's disease	↑	↑/N	N	↑/N	N	N	N	N
Hemophilias	N	↑	N	↑	N	N	N	N
Warfarin toxicity	N	↑	↑	↑	N/↓	N/↓	N/↑	N
Disseminated intravascular coagulopathy	↑	↑	↑	↑	↓	N/↓	↑	↑

ACT, Activated clotting time; APTT, activated partial thromboplastin time; BMBT, buccal mucosa bleeding time; FDP, fibrin degradation products; N, normal; PT, prothrombin time.

Treatment of vitamin K antagonist rodenticide intoxication and other causes of vitamin K deficiency involves supplementation with vitamin K₁ (phytonadione, 5 mg/kg SQ once with 25-gauge needle in multiple sites, and then 2.5 mg/kg PO bid to tid for 30 days). Never administer injections of vitamin K intramuscularly, because of the risk of causing deep muscle hematomas, or intravenously, because of the risk of anaphylaxis. The PT should be rechecked 2 days after the last vitamin K capsule is administered, for some of the second-generation warfarin derivatives are fat-soluble, and treatment may be required for an additional 2 weeks.

Table 1-30 summarizes criteria for interpreting coagulation profiles.

Additional Reading

- Bateman SW, Mathews KA, Abrams-Ogg ACG: Disseminated intravascular coagulation in dogs: a review of the literature, *J Vet Emerg Crit Care* 8:29-45, 1998.
- Bateman SW, Mathews KA, Abrams-Ogg AC, et al: Diagnosis of disseminated intravascular coagulation in dogs admitted to an intensive care unit, *J Am Vet Med Assoc* 215(6):798-804, 1999.
- Bateman SW, Mathews KA, Abrams-Ogg ACG, et al: Evaluation of point-of-care tests for diagnosis of disseminated intravascular coagulation in dogs admitted to an intensive care unit, *J Am Vet Med Assoc* 215:805-810, 1999.
- Couto CG: Spontaneous bleeding disorders. In Bonagura JD, editor: *Current veterinary therapy XII. Small animal practice*, Philadelphia, 1995, WB Saunders.
- Feldman B, Kirby R, Caldin M: Recognition and treatment of disseminated intravascular coagulation. In Bonagura JD, editor: *Kirk's current veterinary therapy XIII*, Philadelphia, 2000, WB Saunders.
- Hackner S: Approach to the diagnosis of bleeding disorders, *Compend Contin Educ Pract Vet* 17:331, 1995.
- Honeckman A, Knapp D, Reagan W: Diagnosis of canine immune-mediated hematologic disease, *Compend Contin Educ Pract Vet* 18:113, 1996.
- Lisciandro SC, Hoenhaus A, Brooks M: Coagulation abnormalities in 22 cats with naturally occurring liver disease, *J Vet Intern Med* 12:71-75, 1998.
- Mischke R, Grebe S, Jacobs C, et al: Amidolytic heparin activity and values for several hemostatic variables after repeated subcutaneous administration of high dose low molecular weight heparin in healthy dogs, *Am J Vet Res* 62(4):595-598, 2001.
- Peterson J, Couto G, Wellman M: Hemostatic disorders in cats: a retrospective study and review of the literature, *J Vet Intern Med* 9:298-303, 1995.
- Stokol T, Brooks MB, Erb HN, et al: D-dimer concentrations in healthy dogs and dogs with disseminated intravascular coagulation, *Am J Vet Res* 61:393-398, 2001.

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BURNS**THERMAL INJURY**

Thermal burns are fortunately a relatively infrequent occurrence in veterinary patients. Box 1-33 lists various causes of malicious and accidental burns. The location of the burn is also important in assessing its severity and potential to lose function. Burns on the perineum, feet, face, and ears are considered to be the most severe because of loss of function and severe pain. Often the severity of thermal injury is difficult to assess in animals because hair coat potentially can mask clinical signs and because the thermal injury can continue after the animal has been removed from the heat source. The skin cools slowly and warms slowly, considerations that become important when initiating therapy for burns. The severity of thermal injury is associated with the temperature to which the animal is exposed, the duration of contact, and the ability of the tissue to dissipate heat. The tissue closest to the heat source undergoes necrosis and has decreased blood flow.

The severity of thermal burn injury is associated directly with the temperature to which the animal is exposed, the percentage of total body surface area affected, the thickness of injured tissue, and whether underlying complications with other body systems occur. Prognosis largely depends on the total body surface area affected (Table 1-31).

Superficial partial thickness, or first-degree, burns offer the most favorable prognosis. The affected epidermis initially appears erythematous and then quickly desquamates within 3 to 6 days. In most cases, fur grows back without leaving a scar. Deep partial thickness, or second-degree, burns involve the epidermis and dermis and are associated with subcutaneous edema, inflammation, and pain. Deep partial thickness burns heal from deeper adnexal tissues and from the wound edges and are associated with an increased chance of scarring and depigmentation. The most severe type is known as full thickness, or third-degree, burns, in which thermal injury destroys the entire thickness of the skin and forms an eschar. Thrombosis of superficial and deeper skin vasculature and gangrene occurs. Treatment involves sequential wound debridement. Healing occurs by second intention and reepithelialization or by wound reconstruction. In most cases, scarring is extensive in affected areas.

Burns greater than 20% of total body surface area will have systemic effects, including impaired cardiovascular function, pulmonary dysfunction, and impaired immune function. Burned tissue, with capillary damage, has increased permeability. The release of inflammatory cytokines, oxygen-derived free radical species, prostaglandins, leukotrienes,

BOX 1-33 CAUSES OF THERMAL INJURY

- Automobile engines
- Automobile exhaust systems
- Boiling water
- Cooking oil (hot)
- Electric heating pads
- Hair dryers
- Heat lamps
- Heat packs
- Improperly grounded electrosurgical units
- Semiliquids (i.e., hot tar)
- Solar exposure
- Steam
- Stove

TABLE 1-31 Percent Burn Estimation: Rule of Nines

Body region	Percent of body surface area
Head	9%
Torso	18%
Forelimb (per limb)	9%
Hind limb (per limb)	18%

histamine, serotonin, and kinins results in increased vascular permeability and leakage of plasma proteins into the interstitium and extravascular space.

Immediate action/treatment

At the time of presentation, first examine the patient and ascertain whether airway obstruction, impaired ventilatory function, circulatory shock, or pain are present. If necessary, establish an airway with endotracheal intubation or emergency tracheostomy. Next, cool the burned area(s) with topical cool water. Use care to avoid overcooling and iatrogenic hypothermia. The best approach is to cool only one portion of the patient's body at a time, then dry, and repeat the process for all affected areas to avoid overcooling and iatrogenic hypothermia. Establish vascular access and administer appropriate and judicious analgesic drugs and intravenous fluid therapy. Whenever possible, avoid placing a catheter through an area of burned or damaged skin. In the early stages of burn injury, shock doses of intravenous crystalloid fluids usually are not required. Later, however, as severe tissue exudation occurs, protein and fluid losses can become extensive, requiring aggressive crystalloid and colloid support to treat hypovolemia and hypoproteinemia. Flush the eyes with sterile saline and examine behind the third eyelids for any particulate matter. Stain the corneas to make sure that superficial corneal burns are not present. Treat superficial corneal burns with triple antibiotic ophthalmic ointment.

Next, assess the total body surface area affected, as this will gauge prognosis. Depending on the extent of the damage, decide whether the burn is superficial and local therapy is indicated or whether more severe injuries exist that may involve systemic therapy or possibly euthanasia.

Differential diagnosis

In most cases the diagnoses of thermal burns are based on a clinical history of being in a house fire, clothes dryer, or under a heating lamp. Too frequently, however, thermal burns become apparent days after an elective surgical procedure in which the patient was placed on a faulty heating pad rather than a circulating warm water or warm air blanket. Superficial burns appear as singed fur with desquamating, easily epilated hair. This condition also can resemble a superficial or deeper dermatophytosis if history is unknown. Other differential diagnoses include immune-mediated vasculitis or erythema multiforme. Unless the superficial dermis is blistered, it may be difficult to distinguish between a thermal burn, chemical burn, or electrical burn if the trauma went unnoticed.

Management

Management of burn injury largely depends on the depth of injury and the total body surface area affected. Partial thickness burns and those affecting less than 15% of the total body surface area will require support in the form of antibiotic ointment and systemic analgesic drugs.

Burns affecting greater than 15% of total body surface area or deep thickness burns require more aggressive therapy. Central venous catheters can be placed to administer crystalloid and colloid fluids, parenteral nutrition if necessary, antibiotics, and analgesic drugs. Monitor perfusion parameters closely, including heart rate, blood pressure, capillary refill time, and urine output. Respiratory function can be impaired because of concurrent smoke inhalation, thermal damage to the upper airways and alveoli, and carboxyhemoglobin or methemoglobin intoxication. Respiratory function also can be impaired because of burn injury to the skin around the thoracic cage. Thoracic radiographs may reveal patchy interstitial to alveolar infiltrates associated with pulmonary edema, pneumonia, and atelectasis. Bronchoscopy often reveals edema, inflammation, particulate matter, and ulceration of the tracheobronchial tree. In some cases, upper airway inflammation is so severe that an emergency tracheostomy must be performed to treat airway obstruction. Administer supplemental humidified oxygen at 50 to 100 mL/kg/minute via endotracheal tube, tracheostomy, nasal or intratracheal tube, or hood oxygen if respiratory function and hypoxemia are present. Perform blood work including a hematocrit, albumin, BUN, creatinine, and glucose at

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the time of presentation. Monitor serum electrolytes, albumin, and colloid oncotic pressure closely because derangements can be severe as burns become exudative.

The goal of fluid therapy in the burn patient is to establish and maintain intravascular and interstitial fluid volume, normalize electrolyte and acid-base status, and maintain serum albumin and oncotic pressure. In the first 24 hours following burn injury, direct fluid therapy to maintaining the patient's metabolic fluid requirements. Crystalloid fluids in the form of Normosol-R, Plasmalyte-M, or lactated Ringer's solution can be administered according to the patient's electrolyte and acid-base status (see Fluid Therapy). Monitor urine output, and keep it at 1 to 2 mL/kg/hour. Avoid overhydration in the early stages of burn injury. In affected burn patients, calculate the amount of fluid that should be administered over a 24-hour period from the formula $1 - 4 \text{ mL/kg} \times \text{percent total body surface area}$. Administer half of this calculated dose over the first 8 hours and then the remaining half over the next 16 hours. In cats, administer only 50% to 75% of this calculated volume. To administer this volume and also avoid fluid overload is often difficult in critically ill patients with pulmonary involvement associated with smoke inhalation injury. Avoid colloids in the first 6 hours after burn injury. Monitor the patient closely for serous nasal discharge, chemosis, and rales that may signify pulmonary edema.

As burns become exudative, weigh the patient at least twice daily. Infused fluid should equal fluid output in the form of urine and wound exudates. Acute weight loss signifies acute fluid loss and that crystalloid fluid infusion should be more aggressive. Ideally, keep the patient's serum albumin equal to or greater than 2.0 g/dL and total protein between 4.0 and 6.5 g/dL using a combination of fresh frozen plasma or concentrated human albumin. Adjunct colloidal support can be provided with synthetic colloids including hetastarch or HBOCs. Keep serum potassium within 3.5 to 4.5 mEq/L using potassium chloride or potassium phosphate supplementation. If potassium supplementation exceeds 80 to 100 mEq/L and the patient continues to have severe refractory hypokalemia, administer magnesium chloride (0.75 mEq/kg/day) to enhance potassium retention. If anemia occurs, administer packed RBCs or whole blood (see Blood Component Therapy).

Lavage wounds daily with lactated Ringer's solution or 0.9% sodium chloride solution. Place wet-to-dry bandages or bandages soaked in silver sulfadiazine or nitrofurazone ointment over the wounds. Depending on the thickness of the burn, epilation and eschar formation and separation may take 2 to 10 days. At each bandage change, debride devitalized tissue to normal tissue. Perform staged partial or total escharectomy, and leave the wound to heal by second intention or by reconstruction using skin advancement flaps or grafts. Maintain meticulous sterility at all times, given that burn patients are at high risk for infection. Administer broad-spectrum antibiotics including cefazolin and enrofloxacin. Perform wound culture if a resistant bacterial infection is suspected.

ELECTRICAL INJURY

The most common cause of electrical injury is associated with an animal chewing on low-voltage alternating current electrical cords in the household. Damage is caused by the current flowing through the path of least resistance, causing heat and thrombosis of vessels and neurons. In some cases, the owner witnesses the event. In other cases, the owner presents the patient because of vague nonspecific signs, and characteristic abnormalities on physical examination support a diagnosis of electrocution. Burns on the face, paws, commissures of the mouth, tongue, and soft palate may be present. Electrocution causes a massive release of catecholamines and can predispose the patient to noncardiogenic pulmonary edema within 36 hours of the incident. Clinical signs may be isolated to the pulmonary system, including orthopnea, pulmonary crackles, and cyanosis.

Immediate action

Assess the patient's lips, tongue, soft palate, gingivae, and commissures of the mouth. Early after electrocution, the wound may appear small and white, black, or yellow. Later, the wound may become larger as tissue sloughs because of damaged vascular supply. Assess the patient's respiratory status. Auscultate the lungs to determine whether pulmonary crackles

are present. If the patient is stable, thoracic radiographs may demonstrate an interstitial to alveolar lung pattern in the dorsocaudal lung fields. Measure the patient's heart rate, blood pressure, oxygenation as determined by pulse oximetry or arterial blood gas and urine output. Immediate treatment consists of judicious use of analgesics for the burn injury, antibiotics (cefazolin, 22 mg/kg q8h; cephalexin, 22 mg/kg q8h), and humidified supplemental oxygen (50 to 100 mL/kg/minute). Direct fluid therapy at providing the patient's metabolic fluid requirements. Because of the risk of development of noncardiogenic pulmonary edema, avoid overzealous administration of crystalloid fluids.

Differential diagnosis

Differential diagnoses for the patient with electrical burn injury and electrocution include chemical or thermal burn, immune-mediated glossitis, cardiogenic pulmonary edema, and pneumonia.

Management

Management of the patient with electrical burn injury and electrocution primarily involves the administration of analgesic agents, supplemental humidified oxygen, and topical treatment of electrical burns. The noncardiogenic pulmonary edema is typically unresponsive to diuretics (i.e., furosemide), bronchodilators (i.e., aminophylline), and splanchnic vascular dilators (i.e., low-dose morphine). The use of glucocorticoids has no proven benefit and may impair respiratory immune function and is therefore contraindicated. Oral burns may require debridement and advancement flaps if large defects or oronasal fistulas develop. If oral injury is severe, place an esophagostomy or percutaneous gastrostomy tube to ensure adequate nutrition during the healing process. If an animal survives the initial electrocution, prognosis is generally favorable with aggressive supportive care.

CHEMICAL INJURY

Chemical burns are associated with a number of inciting causes, including oxidizing agents, reducing agents, corrosive chemicals, protoplasmic poisons, desiccants, and vesicants. The treatment for chemical burns differs slightly from that for thermal burns, so it remains important to investigate the cause of the burn when providing initial treatment, whenever possible. At the scene, advise the owner to wrap the patient in a clean towel for transport. Chilling can be avoided by then wrapping the patient in a second or third blanket. Placement of ointments by well-doers should be avoided. Encourage immediate transport to the nearest triage facility.

Immediate action/treatment

The first and foremost consideration when treating a patient with chemical burn is to remove the animal from the inciting cause or offending agent. Make no attempt to neutralize alkaline or acid substances because the procedure potentially could cause an exothermic reaction, leading to thermal injury in addition to the chemical injury.

Remove collars or leashes that may act as tourniquets or constricting devices. Flush affected areas with copious amounts of cool water for several minutes, not cooling more than 10% to 20% of the body at any one time to prevent iatrogenic hypothermia. Support breathing by extending the patient's head and neck.

Carefully clip the fur over affected areas for further evaluation of the extent of the injury. Lavage exposed eyes with sterile saline, and stain the cornea to evaluate for any corneal burns. Debride any wounds carefully, knowing that the full extent of the wound may not manifest itself for several days. Then cover the wounds with antibiotic burn ointment such as silver sulfadiazine and an occlusive dressing.

Differential diagnosis

Without a history of exposure, the differential diagnosis for any chemical burn includes thermal burn, necrotizing vasculitis, erythema multiforme, or superficial or deep pyoderma.

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Management

Contact local or national animal poison control regarding whether to attempt neutralization. Perform daily bandage changes with staged debridement as the full extent of the wound manifests itself. Place antimicrobial ointment and silver sulfadiazine ointment over the wound to prevent infection. The routine use of antibiotics may promote the development of a resistant bacterial infection. First-generation cephalosporin can be administered. If a more serious infection develops, perform culture and susceptibility testing to direct appropriate antibiotic therapy. The wound can heal by second intention or may require reconstructive repair for definitive closure.

RADIATION INJURY

The primary cause of radiation injury in small animal patients is radiation therapy for neoplastic conditions. The goal of radiation therapy is to kill neoplastic cells. An unfortunate side effect is damage to adjacent normal tissue that results in necrosis, fibrosis, and impaired circulation to the affected area. Radiation burns result in dermatitis, mucositis, impaired surgical wound healing, and chronic nonhealing wounds. In many cases, the degree of secondary radiation injury to normal tissue can be prevented or decreased with careful radiation planning and mapping of the radiation field, such that radiation exposure to normal tissue is limited to the smallest extent possible. With the advent of three-dimensional imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI), this has become more routine in veterinary oncology to date.

Radiation injury can be early and appear at the later stage of the course of radiation therapy. Late effects can be delayed and occur 6 months to years after treatment. The degree of radiation injury is categorized based on the depth of tissue affected. First-degree changes cause cutaneous erythema. Second-degree changes cause superficial desquamation. Third-degree changes cause deeper moist desquamation, and fourth-degree changes are associated with complete dermal destruction and ulceration. During the early stages of radiation injury, affected tissues may appear erythematous and edematous. Wound exudates may be moist, or the skin may appear dry and scaly with desquamation or ulceration. Later, the area may scar and depigment or may have induration, atrophy, telangiectasia, keratosis, and decreased adnexal structures.

Immediate action/treatment

Treatment for radiation dermatitis is to irrigate the area with warmed saline and to protect the area from self-mutilation. No-bite, or Elizabethan, collars or loose clothing can be used to protect the area for patient-induced injury. Mucositis can be treated with topical green tea baths and the administration of an oral solution of L-glutamine powder (4 g/m²). Local irrigation of xylocaine or lidocaine viscous jelly can be used in dogs but should be avoided in cats because of the risk of inducing hemolytic anemia and neurotoxicity. Topical and systemic antibiotics (cephalexin, 22 mg/kg PO tid) also can be administered. Avoid antibiotics that can be sensitized by radiation (i.e., metronidazole).

Differential diagnosis

Because most radiation burns are associated with a known exposure to radiation therapy, the cause of the patient's injury usually is known. If an animal presents to you with a scar, however, differential diagnoses may include nasal planum solar dermatitis, pemphigus foliaceus, discoid lupus, superficial necrolytic dermatitis, superficial or deep pyoderma, chemical burn, or thermal burn.

Management

Treatment of radiation injury involves making the patient as comfortable as possible with analgesic drugs, prevention of self-mutilation, and staged debridement techniques. Wounds can heal by second intention or may require reconstructive surgery.

Additional Reading

- Adamiak Z, Brzeski W, Nowicki M: Burn wound management with hydrocolloid dressings in dogs, *Aust Vet Pract* 32(4):171-172, 2002.
- Aragon CL, Harvey SE, Allen SW, et al: Partial thickness skin grafting for large thermal wounds in dogs, *Compend Contin Educ Pract Vet* 26(3):200-212, 2004.
- Dernell WS, Wheaton LG: Surgical management of radiation injury: part I, *Compend Contin Educ Pract Vet* 17:181, 1995.
- Dernell WS, Wheaton LG: Surgical management of radiation injury: part II, *Compend Contin Educ Pract Vet* 17:499, 1995.
- Papazoglou LG, Kazakos G, Moustardas N: Thermal burns in 2 dogs associated with inadequate grounding of electrosurgical unit patient plates, *Aust Vet Pract* 21(2):67-70, 2001.
- Pope ER, Payne JT: Pathophysiology and treatment of thermal burns. In Harari J, editor: *Surgical complications and wound healing in small animal practice*, Philadelphia, 1993, WB Saunders.
- Singh A, Cullen DL, Grahn BH: Alkali burns to the right eye, *Can Vet J* 45(9):777-778, 2004.

CARDIAC EMERGENCIES

CARDIAC ARREST AND CARDIOPULMONARY CEREBRAL RESUSCITATION

Cardiopulmonary arrest is the abrupt cessation of spontaneous and effective ventilation and perfusion. Cardiac arrest must be treated rapidly and aggressively for any chance of success. The goal of cardiopulmonary cerebral resuscitation (CPCR) is to perform effective thoracic compressions such that an adequate amount of oxygen is delivered to the brain and other vital tissues. At the time of admission into the hospital, all patients, regardless of their disease process, should have a plan in the event that cardiopulmonary arrest occurs. Do the owners want to proceed with CPCR? Should you proceed with intubation, cardiac compressions and drugs, or do the owners want you to perform open-chest CPCR?

One of the most important aspects of cardiopulmonary resuscitation is to anticipate whether a patient is rapidly decompensating and likely to arrest and to be prepared at all times. Stock a crash cart at all times with the equipment and drugs necessary in the event that cardiopulmonary resuscitation is required (Box 1-34).

By having routine drills in the hospital on cadavers or stuffed animals, your emergency team can become efficient at performing the responsibilities and jobs required for successful CPCR. The staff should know how to recognize impending signs of a decompensating patient, clinical signs of cardiac arrest, how to call for an emergency in the hospital, how to intubate patients, and how to start cardiac compressions, hook up an ECG, and draw up the drugs required for various arrhythmias.

Conditions that predispose a patient to cardiopulmonary arrest include vagal stimulation, cellular hypoxia, septicemia, endotoxemia, severe acid-base and electrolyte derangements, prolonged seizures, pneumonia, pleural or pericardial effusion, severe multisystemic trauma, electrical shock, urinary obstruction or damage, acute respiratory

BOX 1-34 ITEMS TO STOCK IN THE CRASH CART

- Laryngoscope (various size blades)
- Endotracheal tubes (various sizes)
- Cotton roll gauze to tie in endotracheal tube
- Stylette for intubation
- Rigid catheter (tomcat and long urinary) to assist with intubation and endotracheal drug administration
- 3-, 6-, and 12-mL syringes, taken out of case and attached to 22-gauge needles
- 22-gauge needles
- Ambubag and oxygen source
- Electrocardiogram monitor
- Epinephrine
- Atropine
- Naloxone
- Calcium gluconate or calcium chloride
- Magnesium chloride
- Amiodarone
- 0.9% saline
- 50% dextrose
- Laceration pack for slash tracheostomy
- Intravenous catheters
- 1-inch white tape
- Emergency drug table with dose and volume and route of administration for various size animals

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distress syndrome (ARDS), and anesthetic agents. The acute onset of bradycardia, change in mucous membrane color and capillary refill time, change in respiratory pattern, and change in mentation are signs of possible deterioration and impending cardiopulmonary arrest.

The diagnosis of cardiopulmonary arrest is based on the absence of effective ventilation, severe cyanosis, absence of a palpable pulse or apex heartbeat, absence of heart sounds, and ECG evidence of asystole or other nonperfusing rhythm such as electrical-mechanical dissociation (aka pulseless electrical activity) or ventricular fibrillation.

Immediate action/treatment*Cardiopulmonary cerebral resuscitation*

The goals of CPR are to obtain airway access, provide artificial ventilation and supplemental oxygen, implement cardiac compressions and cardiovascular support, recognize and treat dysrhythmias and arrhythmias, and provide stabilization and treatment for cardiovascular, pulmonary, and cerebral function in the event of a successful resuscitation. Even with aggressive treatment and management, the overall success of CPR is less than 5% in critically ill or traumatized patients and 20% to 30% in anesthetized patients.

Basic life support

Basic life support involves rapid intubation to gain airway access, artificial ventilation, and cardiac compressions to promote blood flow and delivery of oxygen to the brain and other important tissues (Figure 1-26). Perform the ABCs or CABs of CPR, where *A* is airway, *B* is breathing, and *C* is compression and circulation. Recently, the paradigm has shifted to CABs. While a team member is grabbing an endotracheal tube, clearing the airway of foreign debris, and establishing airway access through endotracheal intubation, a second person starts external cardiac compressions to deliver oxygen that is in the bloodstream to the vital organs. The patient should be positioned in dorsal (> 7 kg) or lateral (< 7 kg) recumbency for external cardiac compressions. Approximately 80 to 120 external compressions should be performed over the patient's sternum. A team member should palpate for a peripheral pulse to determine whether cardiac compressions are actually effective. If a peripheral pulse cannot be palpated for every chest compression, change the patient's position and have a larger individual perform compressions, or initiate open-chest cardiac resuscitation. Once the patient is intubated, tie in the endotracheal tube and attach it to an oxygen source (anesthetic machine or mechanical ventilator or Ambu bag) for artificial ventilation. The oxygen flow rate should be 150 mL/kg/minute. Give two long breaths, and then 12 to 16 breaths per minute. Simultaneous ventilation with thoracic compression increases the pressure difference in the thorax and allows more forward flow of oxygenated blood through the great vessels into the periphery. If possible, a third team member can initiate interposed abdominal compressions, compressing the abdomen when the thoracic cage is relaxed, to improve forward flow. If only one person is available to perform the thoracic compressions and ventilation, give two breaths for every 15 compressions (i.e., 15 thoracic compressions followed by two long breaths, and then start thoracic compressions again). The Jen Chung maneuver can be performed by placing a 25- to 22-gauge hypodermic needle through the skin of the nasal philtrum and twisting the needle into the perosteum to stimulate respirations. This maneuver appears to work better in cats than dogs at return to spontaneous respiration.

Advanced life support

Advanced life support during CPR involves ECG, pulse oximetry and capnometry monitoring, administration of drugs, and the administration of intravenous fluids (in select cases). Most of the drugs used during CPR can be administered directly into the lungs from the endotracheal tube (intratracheal tube). Therefore, only in select instances is it necessary to establish vascular or intraosseous access during CPR (Figure 1-27). If an animal experiences cardiopulmonary arrest because of extreme hemorrhage or hypovolemia,

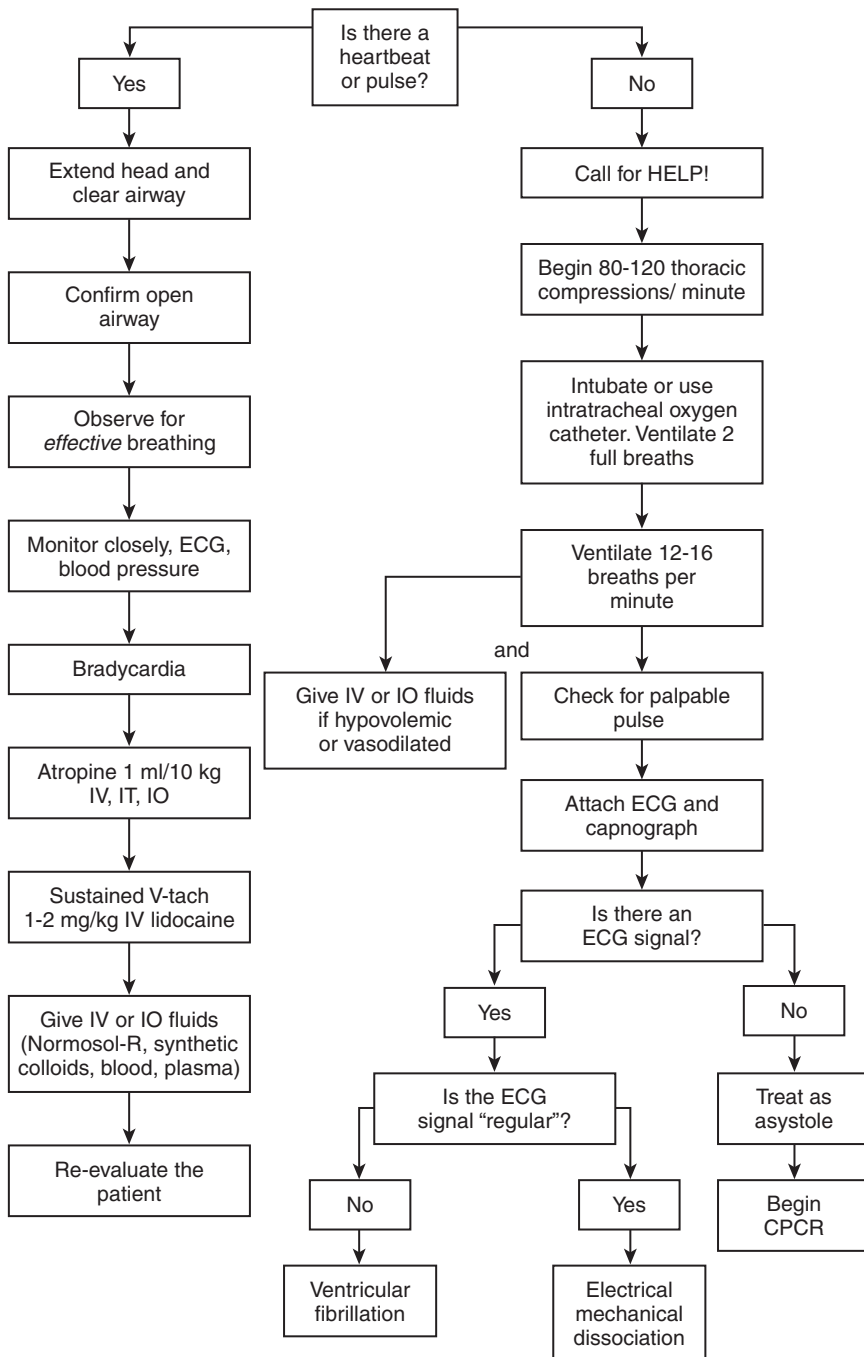


Figure 1-26: Basic cardiopulmonary life support. *ECG*, Electrocardiogram; *CPCR*, cardiopulmonary cerebral resuscitation.

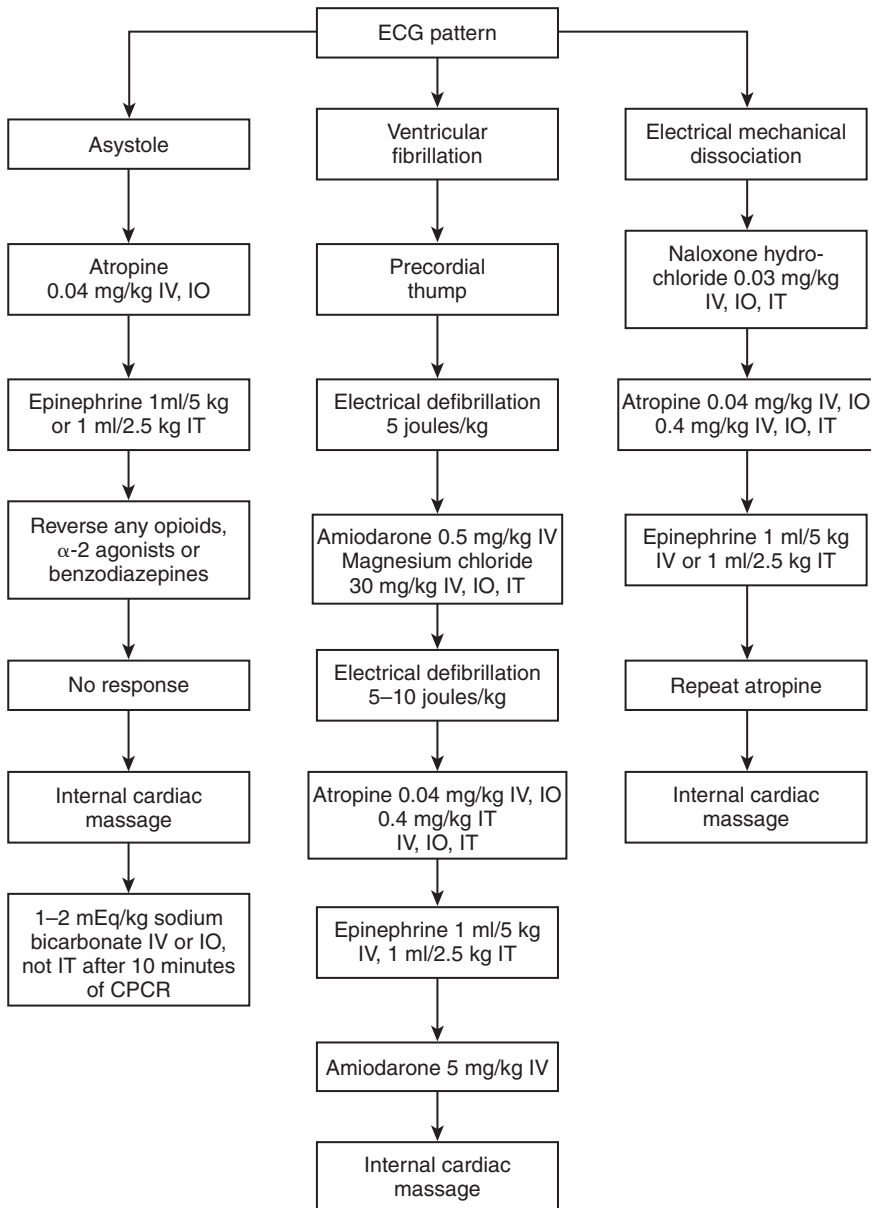


Figure 1-27: Advanced cardiopulmonary life support

inappropriate vasodilation caused by sepsis or systemic inflammation, or vasodilation resulting from anesthesia, the administration of shock volumes (90 mL/kg/hour in dogs and 44 mL/kg/hour in cats) is appropriate. If a patient is euvoletic and experiences cardiopulmonary arrest, however, an increase in circulating fluid volume actually can impair coronary artery perfusion by increasing diastolic arterial blood pressure and is

therefore contraindicated. Place a capnograph on the end or side of the endotracheal tube to measure end-tidal carbon dioxide.

RECOGNITION AND TREATMENT OF COMMON NONPERFUSING CARDIAC RHYTHMS DURING CPR

Asystole: “He’s flatlined”

Asystole is one of the most common rhythm disturbances that causes cardiac arrest in small animal patients. One of the most important things to do when the ECG looks like asystole is to make sure that the ECG monitor is working properly and that all ECG leads are attached properly to the patient. If asystole is truly present, reverse any opiate, α_2 -agonist, or benzodiazepine drugs with their appropriate reversal agents. Low-dose epinephrine (0.02 to 0.04 mg/kg diluted with 5 mL sterile saline) can be administered directly into the endotracheal tube via a rigid or red rubber catheter. If vascular access is available, epinephrine (0.02 to 0.04 mg/kg) can be administered intravenously. No drug should ever be administered directly into the heart by intracardiac injection. Unless the heart is in the veterinarian’s hand during open-chest CPR, intracardiac injection is risky and potentially could lacerate a coronary artery or cause the myocardium to become more irritable and refractory to other therapies, if a drug is delivered into the myocardium and not into the ventricle. For these reasons, intracardiac injections are contraindicated.

Administer atropine (0.4 mg/kg IV, IO, or 0.4 mg/kg IT) immediately after the epinephrine. Atropine, a vagolytic drug, serves to decrease tonic vagal inhibition of the sinoatrial and atrioventricular node and increase heart rate. Administer atropine and epinephrine every 2 to 5 minutes during asystole while cardiac compressions, interposed abdominal compressions, and artificial ventilation are continued. Although discontinuation of thoracic compressions can decrease the chance of success during CPR, you must intermittently evaluate the ECG monitor for any rhythm change that may require different drug therapies. If the cardiac arrest was not witnessed or more than 2 to 5 minutes have passed without successful return to a perfusing rhythm, perform open-chest CPR, if the client wishes. Administer sodium bicarbonate (1 to 2 mEq/kg IV) every 10 to 15 minutes during CPR. Sodium bicarbonate is the only drug used in CPR that **SHOULD NOT** be administered intratracheally because of inactivation of pulmonary surfactant.

Electrical-mechanical dissociation

Electrical-mechanical dissociation also is known as pulseless electrical activity and is an electrical rhythm that may look wide and bizarre and irregular with no associated mechanical contraction of the ventricles. The rhythm can appear different from patient to patient. Electrical-mechanical dissociation is one of the more common nonperfusing rhythms observed during cardiopulmonary arrest in small animal patients (Figure 1-28).

When electrical-mechanical dissociation is identified, first confirm the rhythm and proceed with CPR as previously described. Electrical-mechanical dissociation is thought to be associated with high doses of endogenous endorphins and high vagal tone. The treatment of choice for electrical-mechanical dissociation is high-dose atropine (4 mg/kg IV, IT [10 times the normal dose]) and naloxone hydrochloride (0.03 mg/kg IV, IO, IT). Administer epinephrine (0.02 to 0.04 mg/kg diluted in 5 mL sterile 0.9% saline IT). If the rhythm does not change within 2 minutes, consider open-chest cardiac massage.

Ventricular fibrillation

Ventricular fibrillation can be coarse (Figure 1-29). Patients with coarse ventricular fibrillation are easier to defibrillate than those with fine defibrillation. If ventricular fibrillation is identified, initiate CPR as described previously (Figure 1-30). If an electrical defibrillator is available, administer 5 J/kg of direct current externally. When a patient in cardiopulmonary arrest is attached to ECG leads, it is important to use contact electrode paste, water-soluble gel such as KY jelly, or water, rather than any form of alcohol. Electrical defibrillation of a patient who has alcohol on the ECG leads can lead to fire and thermal burns. Reverse any opioid, α_2 -agonist, and phenothiazine drugs that have been administered to the patient. If fine ventricular fibrillation is identified, administer epinephrine

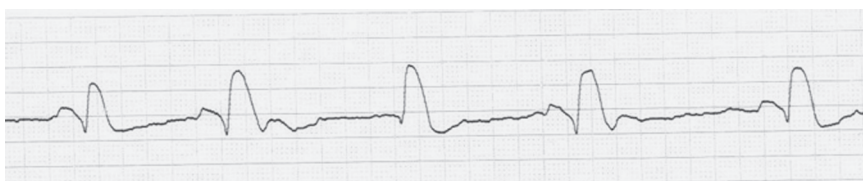


Figure 1-28: Electrical-mechanical dissociation (EMD), also known as pulseless electrical activity (PEA). The complexes often appear wide and bizarre without a palpable apex beat or functional contraction of the heart. This is just one example of EMD, as many shapes and complexes may be observed.

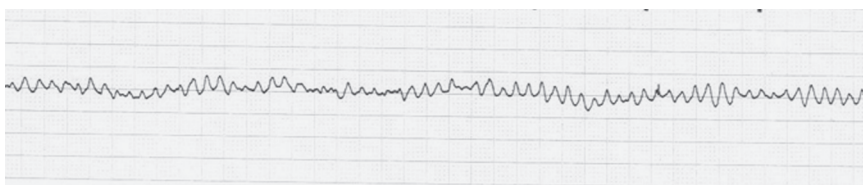


Figure 1-29: Rhythm strip of ventricular fibrillation.

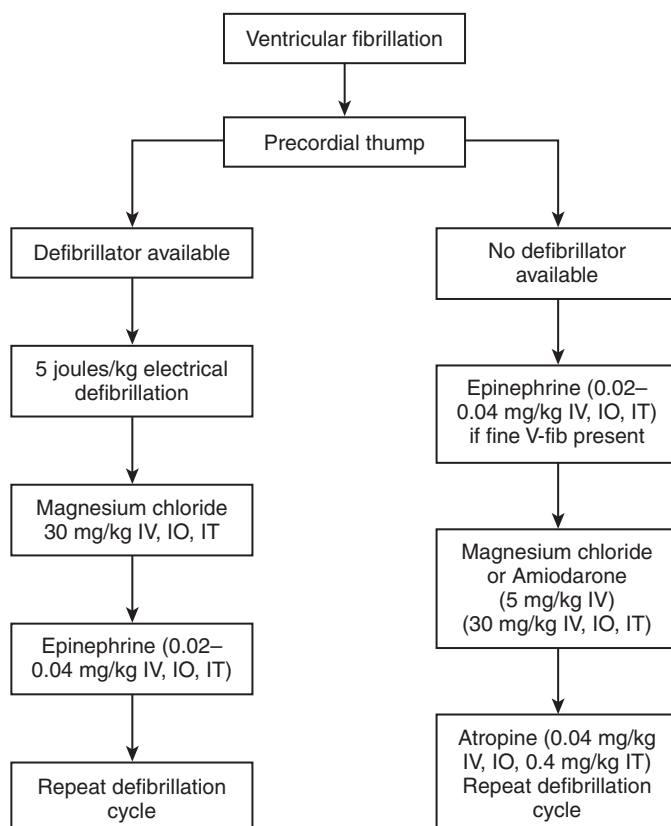


Figure 1-30: Algorithm for treatment of ventricular fibrillation (V-fib). This algorithm is organized according to whether an electrical defibrillator is available. After each intervention step, the ECG should be reevaluated and the next step initiated if V-fib is still seen. If a new arrhythmia develops, the appropriate therapy for that rhythm should be initiated. If a sinus rhythm is seen with a palpable apex beat, postresuscitation measures should be implemented.

BOX 1-35 INDICATIONS FOR IMMEDIATE OPEN-CHEST CARDIOPULMONARY CEREBRAL RESUSCITATION

- Pleural effusion
- Pneumothorax
- Rib fractures or flail chest
- Pericardial effusion
- Diaphragmatic hernia
- Obesity
- More than 5 minutes has passed since cardiopulmonary arrest

(0.02 to 0.04 mg/kg in 5 mL sterile 0.9% saline IT) to attempt to convert fine ventricular fibrillation to coarse ventricular fibrillation. After administration of epinephrine, repeat electrical defibrillation. If an electrical defibrillator is not available, chemical defibrillator drugs can be used. First, administer magnesium chloride (30 mg/kg IV or IT). Even if an electrical defibrillator is available, magnesium chloride can increase the success of converting ventricular fibrillation to asystole or some other rhythm during CPR. Amiodarone (5 mg/kg IV, IO, IT) also can be used to convert ventricular fibrillation. If drug therapy and external thoracic compressions are ineffective after 2 minutes, consider open-chest CPR.

Open-chest cardiopulmonary cerebral resuscitation

Perform open-chest CPR immediately if a pathologic condition exists that prevents enough of a change in intrathoracic pressure that closed-chest CPR will not be effective in promoting forward blood flow (Box 1-35).

To perform open-chest CPR, place the patient in right lateral recumbency. Clip a wide strip of fur over the left fifth to seventh intercostal space and quickly aseptically scrub over the clipped area. Using a No. 10 scalpel blade, incise over the fifth intercostal space through the skin and subcutaneous tissue to the level of the intercostal muscles. With a Mayo scissors, make a blunt stab incision through the intercostal muscles in the left sixth intercostal space. Make sure that the person who is breathing for the patient deflates the lungs as you make the stab incision to avoid iatrogenic lung puncture. After the stab incision, open the tips of the Mayo scissors and quickly open the muscle dorsally and ventrally to the sternum with a sliding motion. Avoid the internal thoracic artery at the sternum and the intercostal arteries at the caudal aspect of each rib. Cut the rib adjacent to the sternum and push it behind the rib in front of and at the caudal aspect of the incision to allow more room and better visualization if a rib spreading retractor is not available. Visualize the heart in the pericardial sac. Visualize the phrenic nerve, and incise the pericardium just ventral to the phrenic nerve. Make sure to not cut the phrenic nerve. Grasp the heart in your hand(s) and gently squeeze it from apex to base, allowing time for the ventricle to fill before the next “contraction.” If the heart does not seem to be filling, administer fluids intravenously or directly into the right atrium. The descending aorta can be cross-clamped with a Rummel tourniquet or red rubber catheter to improve perfusion to the brain and heart.

Management*Postresuscitation care and monitoring (prolonged life support)*

Postresuscitation care involves careful monitoring and management of the adverse effects of hypoxia and reperfusion injury on the brain and other vital organs. The first 4 hours after an arrest are most critical, because this is the time period in which an animal is most likely to rearrest unless the underlying cause of the initial arrest has been determined and treated (Table 1-32). Until an animal is adequately ventilating on its own, artificial ventilation by manual bagging or attaching the patient to a mechanical ventilator with supplemental oxygen must continue. The efficacy of oxygenation and ventilation can be monitored using a Wright’s respirometer, pulse oximetry, capnometry, and arterial blood

TABLE 1-32 Drugs Used in Advanced and Prolonged Life Support

Drug	Dose
Advanced life support	
Atropine	0.04 mg/kg IV, IO; 0.4 mg/kg IT
Amiodarone	5 mg/kg IV, IO, IT
Epinephrine	0.02-0.04 mg/kg IV, IO, IT
Isoproterenol	0.04-0.08 µg/kg/minute IV CRI for third-degree atrioventricular block
Magnesium chloride	30 mg/kg IV, IO, IT
Naloxone	0.03 mg/kg IV, IO, IT
Sodium bicarbonate	1-2 mEq/kg IV, IO; NEVER administer intratracheally
Postresuscitation/Prolonged life support	
Furosemide	1 mg/kg IV
Lidocaine	1-2 mg/kg IV, followed by 50-100 µg/kg/minute CRI
Mannitol	0.51 g/kg IV

gas analyses (see also Pulse Oximetry and Capnometry [End-Tidal Carbon Dioxide Monitoring]). Once an animal is extubated, administer supplemental oxygen (50 to 100 mL/kg/minute) (see Oxygen Supplementation).

The brain is sensitive to ischemia and reperfusion injury. The effects of cellular hypoxia and reperfusion include the development of oxygen-derived free radical species that contribute to cerebral edema. Administer mannitol (0.5 to 1 g/kg IV over 5 to 10 minutes), followed by furosemide (1 mg/kg IV) 20 minutes later, to all patients that have experienced cardiopulmonary arrest and have had successful resuscitation. Mannitol and furosemide work synergistically to decrease cerebral edema formation and scavenge oxygen-derived free radical species.

The combination of cardiac arrest, myocardial ischemia and acidosis, and external or internal cardiac compressions often make the myocardium irritable and predisposed to dysrhythmias following successful CPR. Start lidocaine (1 to 2 mg/kg IV, followed by 50 to 100 µg/kg/minute IV CRI) in all patients following successful resuscitative efforts. Monitor the ECG continuously for the presence of cardiac dysrhythmias and recurrence of nonperfusing rhythms. Perform direct or indirect blood pressure monitoring. If a patient's systolic blood pressure is less than 80 mm Hg, diastolic pressure is less than 40 mm Hg, or mean arterial blood pressure is less than 60 mm Hg, administer positive inotropic drugs (dobutamine, 1 to 20 µg/kg/minute) and pressor agents (epinephrine, 0.02 to 0.04 mg/kg IV, IO, IT) to improve cardiac contractility, cardiac output, and core organ perfusion.

The kidneys are sensitive to decreased perfusion and cellular hypoxia. Place a urinary catheter and monitor urine output. In a euolemic patient, normal urine output should be no less than 1 to 2 mL/kg/hour. If urine output is low, administer low-dose dopamine (3 to 5 µg/kg/minute IV CRI) in an attempt to dilate afferent renal vessels and improve renal perfusion.

Maintain acid-base and electrolyte status within normal reference ranges. Monitor serum lactate as a rough indicator of organ perfusion and cellular oxygen extraction. The presence of elevated or rising serum lactate in the face of aggressive cardiorespiratory and cerebral support makes prognosis less favorable.

Additional Reading

- Cole SG, Otto CM, Hughes D: Cardiopulmonary cerebral resuscitation: a clinical practice review part I, *J Vet Emerg Crit Care* 12(4):261-267, 2002.
 Cole SG, Otto CM, Hughes D: Cardiopulmonary cerebral resuscitation: a clinical practice review part II, *J Vet Emerg Crit Care* 13(1):13-23, 2003.

- Crowe DT: Clinic and staff readiness: the key to successful outcomes in emergency care, *Vet Med* 98(9):760-776, 2003.
- Hackett TB: Cardiopulmonary cerebral resuscitation, *Vet Clin North Am Small Anim Pract* 31(6):1253-1264, 2001.
- Haldane S, Marks SL: Cardiopulmonary cerebral resuscitation: emergency drugs and post-resuscitative care, *Compend Contin Educ Pract Vet* 26(10):791-799, 2004.
- Haldane S, Marks SL: Cardiopulmonary cerebral resuscitation: techniques, *Compend Contin Educ Pract Vet* 26(10):780-790, 2004.
- Johnson T: Use of vasopressin in cardiopulmonary arrest: controversies and promise, *Compend Contin Educ Pract Vet* 25(6):448-451, 2003.
- Kruse-Elliott KT: Cardiopulmonary resuscitation: strategies for maximizing success, *Vet Med* 96(1):51-58, 2001.
- Lehman TL, Manning AM: Post-arrest syndrome and the respiratory and cardiovascular systems in post-arrest patients, *Compend Contin Educ Pract Vet* 25(7):492-502, 2003.
- Lehman TL, Manning AM: Renal, central nervous and gastrointestinal systems in post-arrest patients, *Compend Contin Educ Pract Vet* 25(7): 504-512, 2003.
- Rieser T: Cardiopulmonary resuscitation, *Clin Tech Small Anim Pract* 15(2):76-81, 2000.
- Waldrop JE, Rozanski EA, Swanke Ed, et al: Causes of cardiopulmonary arrest, resuscitation management, and functional outcome in dogs and cats surviving cardiopulmonary arrest, *J Vet Emerg Crit Care* 14(1):22-29, 2004.
- Wingfield WE: Cardiopulmonary arrest. In Wingfield WE, Raffee MR, editors: *The veterinary ICU book*, Jackson, Wyo, 2001, Teton NewMedia.
- Wingfield WE: Cardiopulmonary arrest and resuscitation in small animals. In Wingfield WE, editor: *Veterinary emergency medicine secrets*, ed 2, Philadelphia, 2001, Hanley & Belfus.

CARDIAC DYSRHYTHMIAS REQUIRING EMERGENCY MANAGEMENT

Cardiac dysrhythmias can encompass a wide range of clinical syndromes that vary in their clinical significance and signs, depending on the rate and frequency and whether coexisting cardiac disease is present. Ventricular and supraventricular dysrhythmias can occur because of primary myocardial disease or some other, secondary underlying disease process, including thoracic trauma, sepsis, systemic inflammatory response syndrome, pancreatitis, GDV, splenic disease, hypoxia, uremia, and acid-base and electrolyte disturbances. Common cardiac causes of dysrhythmias include dilative cardiomyopathy, end-stage degenerative valvular disease, infectious endocarditis, myocarditis, and cardiac neoplasia. In the cat, hypertrophy, restrictive, and unclassified cardiomyopathies and hyperthyroidism are the most common causes of dysrhythmias. In addition to structural cardiac or systemic disease, dysrhythmias can occur as an adverse effect of some drugs, including digoxin, dobutamine, aminophylline, and anesthetic agents.

Immediate action

Immediate action depends largely on recognition of the primary or secondary cause of the dysrhythmia and treating the dysrhythmia and underlying cause.

Differential diagnosis

Diagnosis of cardiac dysrhythmias is based on physical examination findings of abnormal thoracic/cardiac auscultation, the presence of abnormal pulse rhythm and quality, and recognition of ECG abnormalities. The ECG is critical to the accurate diagnosis of dysrhythmias.

Ventricular dysrhythmias

Ventricular dysrhythmias arise from ectopic foci in the ventricles that cause the wave of depolarization to spread from cell to cell rather than spread through fast-conducting tissue. This causes the QRS complex to appear wide and bizarre, unless the ectopic focus originates close to the atrioventricular node high in the ventricle. Other ECG features of ventricular dysrhythmias include a T wave polarity that is opposite to the QRS complex and nonrelated P waves. Ventricular dysrhythmias may manifest as isolated ventricular premature complexes, couplets, or triplets; bigeminy; or ventricular tachycardia. Relatively slow ventricular tachycardia is known as an idioventricular rhythm and is not as

1



Figure 1-31: Unifocal premature ventricular complexes (PVCs). All the PVCs are the same shape and size and originate from the same ectopic focus in the ventricle. Note that this rhythm is actually an example of ventricular bigeminy.

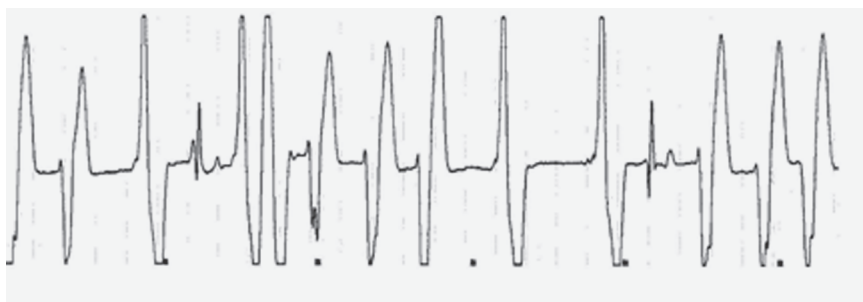


Figure 1-32: Multifocal premature ventricular complexes (PVCs). Note that the complexes change shape, size, and orientation, indicating multiple ectopic foci within the ventricle.

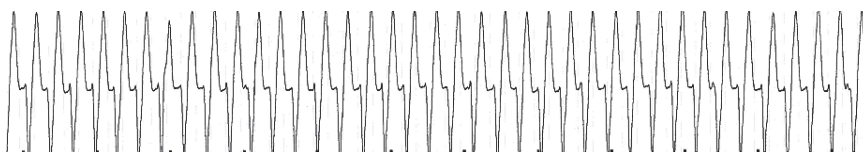


Figure 1-33: Sustained ventricular tachycardia.

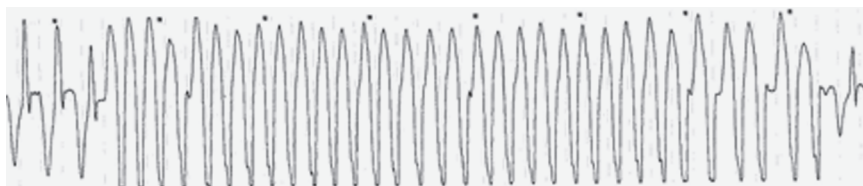


Figure 1-34: An example of R-on-T phenomenon. Note that there is no return to baseline or isoelectric shelf in between the T wave of one complex and the R wave of the next complex. This rhythm can be very dangerous and can lead to ventricular fibrillation.

hemodynamically significant as faster ventricular tachycardia. Idioventricular rhythm usually is less than 130 beats per minute and may alternate spontaneously with sinus arrhythmias (Figures 1-31 to 1-34).

Supraventricular dysrhythmias

Supraventricular dysrhythmias arise from ectopic foci in the atria and are commonly associated with atrial dilatation and structural heart disease such as advanced acquired or congenital heart disease, cardiomyopathies, cardiac neoplasia, or advanced heartworm disease. Occasionally, supraventricular dysrhythmias may be associated with respiratory or other systemic illness. Sustained supraventricular tachycardia in the absence of underlying structural heart or systemic disease is disturbing and should alert the clinician that an accessory pathway conduction disturbance may be present, particularly in Labrador Retrievers.

Supraventricular dysrhythmias can manifest as isolated premature complexes (atrial premature complexes or contractions), sustained or paroxysmal supraventricular tachycardia (atrial tachycardia), or atrial fibrillation or flutter. In the dog, atrial fibrillation most commonly is associated with dilative cardiomyopathy. Rarely and primarily in giant breed dogs, lone atrial fibrillation can occur with no underlying heart disease. Atrial fibrillation and the resultant sustained elevation in ventricular rate are presumed to progress to dilative cardiomyopathy in such breeds. By comparison, atrial fibrillation is relatively uncommon in cats because of the small size of their atria but is associated most commonly with hypertrophic and restrictive cardiomyopathy.

The ECG is critical to the diagnosis of a supraventricular dysrhythmia. The ECG usually demonstrates a normal appearance to the QRS complex unless aberrant conduction occurs in the ventricles, in which case the QRS can be wide but still originate from above the atrioventricular node. In most cases of a supraventricular dysrhythmia, some evidence of atrial activity including P waves, atrial flutter, or atrial fibrillation is apparent. In some cases, it may be difficult to diagnose the exact rhythm without slowing the rate down mechanically or through pharmacologic intervention. Once a rhythm diagnosis is made, appropriate treatment strategies can be implemented (Figures 1-35 and 1-36).

Management

Ventricular dysrhythmias

Treatment of ventricular dysrhythmias largely depends on the number of ectopic foci discharging, the rate and character of the dysrhythmia, and whether the presence of the abnormal beats is of adverse hemodynamic consequence, including risk of sudden death. Many ventricular dysrhythmias, including slow idioventricular rhythms, ventricular bigeminy, or intermittent ventricular premature complexes, do not warrant antiarrhythmic

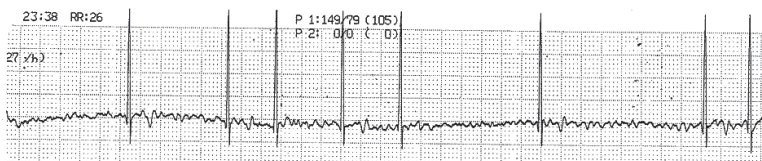


Figure 1-35: Atrial fibrillation.

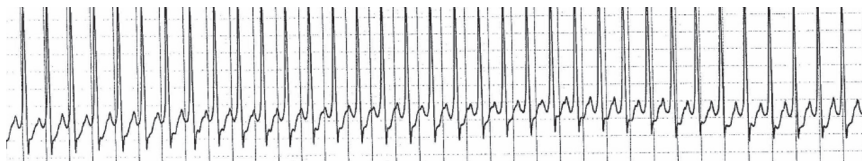


Figure 1-36: Supraventricular tachycardia.

TABLE 1-33 Oral Management of Ventricular Dysrhythmias in Dogs

Drug	Dose
Procainamide	10-20 mg/kg PO q6-8h
Tocainide*	10-20 mg/kg PO q8h
Sotalol	40-120 mg per dog q12h (start low, then titrate up to effect)
Mexiletine	5-8 mg/kg PO q8h
Atenolol	0.25-1.0 mg/kg PO q12-24h (start low, titrate upward to effect)

*Do not use for longer than 2 weeks because of idiosyncratic blindness.

therapy unless the patient is hypotensive and the dysrhythmia is thought to be contributing to the hypotension. In such cases, correction of the underlying disease process including hypoxia, pain, or anxiety often alleviates or decreases the incidence of the dysrhythmia.

More serious ventricular dysrhythmias that warrant antiarrhythmic therapy (Table 1-33) include sustained ventricular tachycardia (>160 beats/minute in dogs; >220 beats/minute in cats), multifocal ventricular premature complexes originating from more than one place in the ventricles, and the presence of R-on-T phenomena where the T wave of the preceding complex is superimposed on the QRS of the next complex with no return to isoelectric shelf in between complexes. Treat these ventricular dysrhythmias immediately and aggressively. In dogs, the mainstay of emergency treatment for ventricular dysrhythmias is lidocaine therapy. Administer lidocaine (1 to 2 mg/kg IV bolus) over a period of 5 minutes to prevent the adverse side effects of seizures or vomiting. The bolus can be repeated an additional 3 times (total dose 8 mg/kg) over 15 minutes, or the patient can be placed on a constant rate infusion (50 to 100 μ g/kg/minute) if control of ventricular tachycardia is accomplished. Also correct the patient's magnesium and potassium deficiencies to maximize the success of lidocaine therapy in the treatment of ventricular tachycardia. Procainamide (4 mg/kg IV slowly over 3 to 5 minutes) also can be used to control ventricular tachycardia. If procainamide is successful at controlling ventricular tachycardia, administer it as a constant rate infusion (25 to 40 μ g/kg/minute). Side effects of procainamide include vomiting, diarrhea, and hypotension.

Chronic oral therapy may or may not be necessary in the treatment of acute ventricular tachycardia. The decision to continue antiarrhythmic therapy depends on the underlying disease process and the expectation of persistent arrhythmogenesis of the underlying disease process. Oral antiarrhythmic therapy is warranted in cases in which a serious ventricular dysrhythmia is recognized but the animal does not require hospitalization, such as the syncopal Boxer with intermittent ventricular dysrhythmias and no evidence of structural heart disease. It deserves emphasis that asymptomatic, low-grade ventricular dysrhythmias probably do not require treatment. If maintenance therapy for ventricular dysrhythmias is needed, use an oral drug based on the underlying disease process, clinical familiarity, class of drug, dosing frequency, owner compliance, concurrent medications, cost, and potential adverse side effects.

Treatment of ventricular dysrhythmias in cats

In the cat the mainstay of antiarrhythmic therapy is the use of a β -adrenergic antagonist. In the acute management of ventricular dysrhythmias in cases of hypertrophic, restrictive, or unclassified cardiomyopathies, consider using injectable esmolol (0.05 to 1.0 mg/kg IV slowly to effect) or propranolol (0.02 to 0.06 mg/kg IV slowly to effect), particularly if the dysrhythmia results from hyperthyroidism. For chronic oral ventricular antiarrhythmic therapy in cats, propranolol (2.5 to 5.0 mg PO per cat q8h) or atenolol (6.25 to 12.5 mg PO per cat q12-24h) can be used.

Supraventricular dysrhythmias

The decision to treat supraventricular dysrhythmias depends on the ventricular rate and the hemodynamic consequences of the dysrhythmia. For intermittent isolated atrial

TABLE 1-34 Parenteral and Oral Management of Supraventricular Dysrhythmias

Drug	Dose
Parenteral	
Esmolol	50-100 µg/kg IV bolus, 50-200 µg/kg/minute IV CRI
Propranolol	0.04-0.1 mg/kg IV slowly to effect
Diltiazem	0.1-0.25 mg/kg IV slowly to effect, then 2-6 µg/kg/minute CRI
Digoxin	0.0025 mg/kg bolus IV; can be repeated every hour up to 0.01 mg/kg maximum dose
Oral	
Digoxin	0.005-0.01 mg/kg PO bid; animal >15 kg, 0.22 mg/m ² PO bid
Diltiazem	0.5 mg/kg PO bid
Diltiazem (Dilacor-XR)	1.5-6 mg/kg q12-24h (dog); cat, 30-60 mg PO q12-24h
Atenolol	0.25-1 mg/kg q12-24h; cat, 6.25 mg PO q12-24h
Propranolol	0.1-0.2 mg/kg PO q8h, titrated up to a maximum of 0.5 mg/kg PO q8h; cat, 2.5-10 mg/kg PO q8h
Amiodarone	10 mg/kg PO q12h for 7 days, then 5 mg/kg PO q24h (maintenance)

premature contractions, couplets, and triplets, usually no treatment is required. When the ventricular rate exceeds 180 beats/minute, diastolic filling time is shortened, causing the heart to not fill adequately. The consequence is decreased cardiac output and decreased coronary artery perfusion. The goal of therapy is rhythm control or, in most cases, rate control. In cases of atrial fibrillation and congestive heart failure, conversion to a normal sinus rhythm rarely can be achieved, although electrocardioversion or pharmacoeversion can be attempted.

In the dog a vagal maneuver can be attempted by pressing on the eyeballs or massaging the carotid body. For sustained supraventricular tachycardia, diltiazem (0.25 mg/kg IV), esmolol (0.05 to 0.1, titrated upward to a cumulative dose of 0.5 mg/kg IV), or propranolol (0.04 to 0.1 mg/kg IV slowly to effect) can be administered in an attempt to slow the ventricular rate in emergent situations. Administer oral diltiazem (0.5 mg/kg PO q8h), diltiazem (Dilacor-XR) (1.5 to 6 mg/kg PO q12-24h), propranolol (0.1 to 0.2 mg/kg tid, titrated up to a maximum of 0.5 mg/kg PO q8h), atenolol (0.25 to 1 mg/kg q12-24h), or digoxin (0.005 to 0.01 mg/kg bid or 0.22 mg/m² for dogs greater than 15 kg).

In the cat a vagal maneuver can be attempted by ocular or carotid massage. (Diltiazem [Dilacor] 30 to 60 PO q12-24h), propranolol (2.5 to 10 mg/kg q12-24h), or atenolol (6.25 mg q12-24h) also can be administered. If structural heart disease is present, treat pulmonary edema and start angiotensin-converting enzyme inhibitor therapy. Table 1-34 summarizes the drugs used in the management of supraventricular dysrhythmias.

BRADYARRHYTHMIAS

Severe bradycardia often results from systemic disease, drug therapy, anesthetic agents, or hypothermia and thus rarely requires specific therapy except to treat or reverse the underlying mechanisms promoting bradycardia. Hemodynamically significant bradyarrhythmias that must be treated include atrial standstill, atrioventricular block, and sick sinus syndrome.

Atrial standstill

Atrial standstill most commonly is associated with hyperkalemia and is seen most often in urinary obstruction, renal failure, urinary trauma with uroabdomen, and hypoadrenocorticism. Characteristic ECG abnormalities observed in atrial standstill are an absence of P waves, widened QRS complexes, and tall spiked T waves (Figure 1-37).

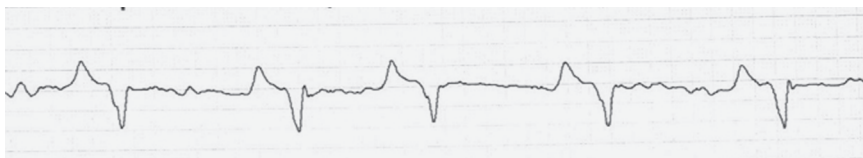


Figure 1-37: An example of atrial standstill caused by hyperkalemia in a blocked tomcat. Note that there are no P waves and that the ventricular QRS complexes are widened and blunted.

The treatment for hyperkalemia-induced atrial standstill is to correct the underlying cause and to drive potassium intracellularly and protect the myocardium from the adverse effects of hyperkalemia. Regular insulin (0.25 to 0.5 units/kg IV) followed by dextrose (1 g/unit insulin IV, followed by 2.5% dextrose CRI to prevent hypoglycemia) or sodium bicarbonate (1 mEq/kg IV) can be administered to drive potassium intracellularly. Calcium gluconate (0.5 mL/kg of 20% solution IV over 5 minutes) also can be administered as a cardioprotective drug until the cause of hyperkalemia has been identified and resolved. Also administer sodium chloride fluids (0.9% sodium chloride IV) to promote kaliuresis.

Less commonly, atrial standstill is associated with atrial cardiomyopathy or silent atrium syndrome. Persistent atrial standstill has been recognized without electrolyte abnormalities in the English Springer Spaniel and the Siamese cat. Short-term therapy for persistent atrial standstill includes atropine (0.04 mg/kg SQ) until definitive treatment by implantation of a cardiac pacemaker can be performed.

Third-degree atrioventricular block

Complete or third-degree atrioventricular block or high-grade symptomatic second-degree atrioventricular block can be hemodynamically significant when ventricular rates are less than 60 beats/minute in the dog. Classic clinical signs include weakness, exercise intolerance, lethargy, anorexia, syncope, and occasionally seizures. Advanced atrioventricular block usually is caused by advanced idiopathic degeneration of the atrioventricular node. Less commonly, atrioventricular block has been associated with digoxin toxicity, magnesium oversupplementation, cardiomyopathy, endocarditis, or infectious myocarditis (Lyme disease). An accurate diagnosis is made based on the ECG findings of nonconducted P waves with ventricular escape beats. First- and second-degree atrioventricular block may not be hemodynamically significant and therefore may not require therapy.

Initially treat third-degree (complete) or symptomatic high-grade second-degree atrioventricular block (<60 beats/minute) with atropine (0.04 mg/kg SQ or IM). Perform a follow-up ECG in 15 to 20 minutes. Atropine is rarely successful in treating complete atrioventricular block. Also attempt treatment with isoproterenol (0.04 to 0.08 µg/kg/minute IV CRI or 0.4 mg in 250 mL 5% dextrose in water IV slowly), a pure β-agonist. Definitive treatment requires permanent pacemaker implantation. Consultation with a veterinary cardiologist who implants pacemakers is suggested. Never attempt to convert or treat the observed ventricular escape beats with lidocaine (Figure 1-38).

Sick sinus syndrome

Sick sinus syndrome most commonly is recognized in the Miniature Schnauzer, although any dog can be affected. Sick sinus syndrome usually results from idiopathic degeneration of the sinus node in the dog. In the cat, sinus node degeneration usually is associated with cardiomyopathy. Dysfunction of the sinus node may manifest as marked bradycardia with periods of sinus arrest followed by junctional or ventricular escape complexes. A variant of sick sinus syndrome is the presence of severe bradycardia followed by periods of supra-ventricular tachycardia, often termed *bradycardia-tachycardia syndrome*. The most common clinical signs are syncope, exercise intolerance, and lethargy.

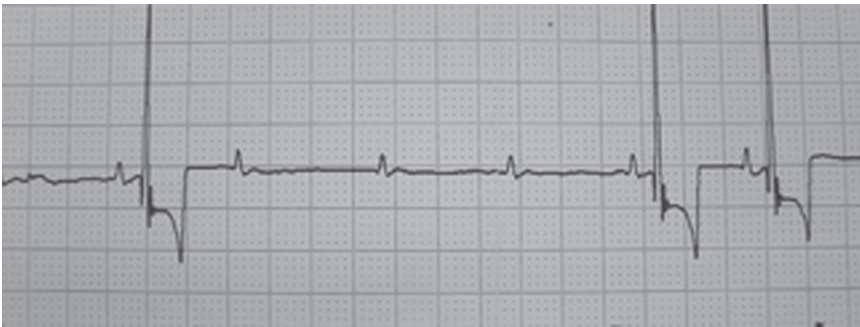


Figure 1-38: Example of third-degree atrioventricular block. Note that there does not appear to be conduction of any of the p-waves, leading to intermittent narrow complex ventricular escape beats.

Treatment of sick sinus syndrome involves permanent pacemaker implantation by a veterinary cardiologist. Less severe cases of sick sinus syndrome can be managed medically, at least short-term, with atropine (0.04 mg/kg IM) or probanthine (0.5 to 1.5 mg/kg PO q8h).

Additional Reading

- Abbott JA: Beta-blockage in the management of systolic dysfunction, *Vet Clin North Am Small Anim Pract* 34(5):1157-1170, 2004.
- Geltzer ARM, Kraus MS: Management of atrial fibrillation, *Vet Clin North Am Small Anim Pract* 34(5):1127-1144, 2004.
- Kittleson MD, Kienle RD: Diagnosis and treatment of arrhythmias. In *Small animal cardiovascular medicine*, St Louis, 1999, Mosby.
- O'Grady MR, O'Sullivan ML: Dilated cardiomyopathy: an update, *Vet Clin North Am Small Anim Pract* 34(5):1187-1207, 2004.
- Wright KN: Interventional catheterization for tachyarrhythmias, *Vet Clin North Am Small Anim Pract* 34(5):1171-1185, 2004.

CONGESTIVE HEART FAILURE IN DOGS AND CATS

Presentation in the dog

The majority of animals that present with congestive heart failure (CHF) are older animals that have some acquired heart disease that develops later in life. Congenital defects are rarer than acquired heart disease. The most common congenital defect observed in dogs and in some cats is a patent ductus arteriosus.

The most common acquired cardiac disease in dogs is chronic valvular disease, or endocardiosis (mitral valve endocardiosis). In endocardiosis, the atrioventricular valves chronically lose the ability to close effectively, causing abnormalities in blood flow, including regurgitation during ventricular systole. In most cases, disease progression is chronic and slow, although acute exacerbations and onset of clinical signs can be associated with stress, rupture of a chordae tendinae, or ingestion of a high-salt meal. Mitral valve disease tends to affect older toy breeds such as miniature Poodles, Chihuahuas, and younger Cavalier King Charles Spaniels.

The second most common cause of acquired heart disease is dilated cardiomyopathy, which is a disease of primary myocardial failure. In dilated cardiomyopathy the muscular wall of the heart becomes thin and weak as the myocardium dilates, causing a decrease in contractility and cardiac output. Secondary mitral and tricuspid valvular insufficiency may result from chronic stretching of the valve annulus. This type of heart disease typically is associated with giant breed dogs including Irish Wolfhounds, English Mastiffs, Great Danes, Boxers, and Doberman Pinschers. A rare form of the disease has been documented in young Labrador Retrievers. Acute exacerbation of dilated cardiomyopathy may be related to the development of a dysrhythmia, including atrial fibrillation.

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Presentation in the cat

In cats, hypertrophic cardiomyopathy is the most common form of acquired cardiac disease observed. Congestive heart failure resulting from hypertrophic cardiomyopathy can occur in animals as young as 6 to 10 months of age. Hypertrophic cardiomyopathy is characterized by stiff, noncompliant ventricles that do not relax during diastole, causing an increase in left atrial pressures and left atrial enlargement. Other cardiomyopathies, including unclassified, restrictive, and dilated, are less common but also can occur in the cat. Cats often develop acute exacerbation of clinical signs because of stress or arterial embolization.

Immediate action/treatment

The rapid diagnosis of CHF often is made on owner history, signalment, and physical examination findings (Box 1-36).

Typical physical examination findings include a cardiac murmur or gallop dysrhythmia, abnormal breath sounds, respiratory difficulty and orthopnea, tachycardia, weak pulse quality, cool peripheral extremities, and pale or cyanotic mucous membrane. Initiate immediate treatment based on physical examination findings and index of suspicion. In some cases, it is difficult to distinguish between CHF and feline lower airway disease (asthma) without performing thoracic radiographs. Let the animal rest and become stabilized before attempting any stressful procedures, including thoracic radiographs.

Immediate treatment consists of administering supplemental oxygen, decreasing circulating fluid volume with furosemide, dilating pulmonary and splanchnic capacitance vessels with topical nitroglycerine and morphine, and alleviating patient anxiety and stress (Box 1-37).

Differential diagnosis

Primary differential diagnoses are made based primarily on the patient's breed, age, clinical signs, history, and physical examination abnormalities. The most common differential diagnoses in a patient with CHF are cardiac abnormalities and respiratory disease (chronic bronchitis [asthma], pulmonary hypertension, cor pulmonale, neoplasia).

Postpone diagnostic tests in any patient with suspected CHF until the immediate treatments have taken effect and the patient is cardiovascularly more stable. In most cases, lateral and dorsoventral thoracic radiographs are one of the most important diagnostic tools in helping make a diagnosis of CHF. Increased perihilar interstitial to alveolar infiltrates are characteristic of pulmonary edema. Left atrial enlargement may be observed as a "backpack" sign at the caudal cardiac waist. Cardiomegaly of the right or left side also may

**BOX 1-36 COMMON PRESENTING COMPLAINTS BY OWNERS
OF PATIENTS WITH CONGESTIVE HEART FAILURE**

- Lethargy
- Weakness
- Cough
- Respiratory difficulty
- Exercise intolerance
- Inappetence
- Weight loss
- Abdominal distention
- Syncope

BOX 1-37 IMMEDIATE MANAGEMENT OF CONGESTIVE HEART FAILURE

- Supplemental oxygen at 50 to 100 mL/kg/minute to supply 40% to 50% oxygen
- Furosemide, 4 to 8 mg/kg IV, IM, every 30 minutes until the patient urinates and body weight decreases by 7%
- Nitroglycerine ointment (1/4 to 1 inch topically) every 8 hours
- Morphine, 0.025 to 0.05 mg/kg IV (dog only)

BOX 1-38 VERTEBRAL HEART SUM TO DETERMINE CARDIOMEGALY**1**

The vertebral heart sum can be calculated by performing the following steps:

1. Measure the long axis of the heart from the apex to the carina on the lateral view and mark the distance on a sheet of paper.
2. Measure the length of the long axis of the heart in terms of vertebral bodies, starting by counting caudally from the fourth thoracic vertebra; count the number of vertebrae that are covered by the length of the long axis of the heart.
3. Measure the short axis of the heart at the caudal vena cava, perpendicular to the long axis of the heart.
4. Count the number of thoracic vertebrae covered by the short axis of the heart, starting at T4.
5. Add the two numbers together to yield the vertebral heart sum; a vertebral heart sum greater than 10.5 is consistent with cardiomegaly.

be present in cases of valvular insufficiency. In cats, increased sternal contact and a classic valentine-shaped heart may be observed in cases of hypertrophic cardiomyopathy. Perform a vertebral heart score (sum) to measure cardiac size and determine whether cardiomegaly is present (Box 1-38).

Also obtain arterial blood pressure and ECG readings to determine whether hypotension and dysrhythmias are present. Atrial fibrillation, ventricular premature contractions, and supraventricular tachycardia are common rhythm disturbances that can affect cardiac output adversely and influence treatment choices.

The echocardiogram is a useful noninvasive and nonstressful method to determine the degree of cardiac disease present. The echocardiogram is largely user-dependent. The quality of the study is based on the experience of the operator and the quality of the ultrasound machine. Echocardiography can be a useful tool in making a diagnosis of pericardial effusion, dilated or hypertrophic cardiomyopathy, cardiac neoplasia, and endocarditis.

Management of congestive heart failure in dogs and cats

The medical management of CHF is designed to improve cardiac output and relieve clinical signs. The immediate goal of therapy is to reduce abnormal fluid accumulation and provide adequate cardiac output by increasing contractility, decreasing preload and ventricular afterload, and/or normalizing cardiac dysrhythmias. Strict cage rest is of utmost importance when managing a patient with CHF.

After initial administration of furosemide, morphine, oxygen, and nitroglycerine paste, clinical signs of respiratory distress should show improvement within 30 minutes. If no improvement is observed, administer repeated doses of furosemide. Reevaluate severe cases that are refractory to this standard treatment protocol. Vasodilation should be the next step in the management of refractory cases, provided that a normal blood pressure is present. Sodium nitroprusside is a potent balanced vasodilator that should be administered (1 to 10 $\mu\text{g/kg/minute}$ IV CRI), taking care to monitor blood pressure continuously because severe vasodilation and hypotension can occur. The goal of nitroprusside therapy is to maintain a mean arterial blood pressure of 60 mm Hg. Sodium nitroprusside should not be considered in cases of refractory CHF with severe hypotension.

For more long-term management of CHF, the use of angiotensin-converting enzyme (ACE) inhibitors including enalapril (0.5 mg/kg PO q12-24h), benazepril (0.5 mg/kg PO q24h), and lisinopril (0.5 mg/kg PO q24h) have become the mainstay of therapy to reduce sodium and fluid retention and decrease afterload. Start angiotensin-converting enzyme inhibition as soon as a patient is able to tolerate oral medications.

Dobutamine (2.5 to 10 $\mu\text{g/kg/minute}$ CRI diluted in 5% dextrose in water) can be administered to improve cardiac contractility, particularly in cases of dilated cardiomyopathy. At low doses, dobutamine, primarily a β -adrenergic agonist, will improve cardiac output with minimal effects on heart rate. Dobutamine must be given as a constant rate infusion with careful, continuous ECG monitoring. Despite minimal effects on heart rate,

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sinus tachycardia or ventricular dysrhythmias may develop during infusion. Cats are more sensitive to the effects of dobutamine than dogs. Monitor carefully for seizures and facial twitching.

Digoxin is a cardiac glycoside that acts as a positive inotrope and negative chronotrope in the long-term management of CHF. Digoxin has a long (24 hours in dogs, and 60 hours in cats) half-life and so has minimal use in the emergency management of CHF. In chronic management of CHF resulting from dilated cardiomyopathy or advanced mitral disease, however, digoxin is extremely useful. Oral digitalization protocols have been developed but are risky in that dysrhythmias and severe gastrointestinal side effects can occur.

Cats with CHF often have fulminant pulmonary edema, pleural effusion, arterial thromboembolism, or some combination of all three. If the pleural effusion is significant, perform therapeutic thoracocentesis to relieve pulmonary atelectasis and improve oxygenation. Once the diagnosis and initial management of CHF has been made, formulate a plan for continued management and monitoring. Tailor the therapeutic plan to the patient based on the cause of the CHF, the presence of concurrent diseases, and response to therapy. An important and often overlooked part of the successful emergency management of CHF is the open communication with the owner regarding the owner's emotional and financial commitment for immediate and long-term management to ensure appropriate quality of life for each patient.

Additional Reading

- Abbott JA: Dilated cardiomyopathy. In Wingfield WE, editor: *Veterinary emergency medicine secrets*, Philadelphia, 2001, Hanley & Belfus.
- Abbott JA: Feline myocardial disease. In Wingfield WE, editor: *Veterinary emergency medicine secrets*, Philadelphia, 2001, Hanley & Belfus.
- Borgarelli M, Tarducci A, Tidholm A, et al: Canine idiopathic dilated cardiomyopathy. 2. Pathophysiology and treatment, *Vet J* 162(3):182-195, 2001.
- Buston R: Treatment of congestive heart failure, *J Small Anim Pract* 44(11):516, 2003.
- Fuentes VL: Use of pimobendan in the management of heart failure, *Vet Clin North Am Small Anim Pract* 34(5):1145-1155, 2004.
- Goodwin JK, Strickland K: The emergency management of dogs and cats with congestive heart failure, *Vet Med* 93(9):818-822, 1998.
- Goodwin JK, Strickland KN: Managing arrhythmias in dogs and cats with congestive heart failure, *Vet Med* 93(9):823-829, 1998.
- Laste NJ: Cardiovascular pharmacotherapy, *Vet Clin North Am Small Anim* 31(6):1231-1252, 2001.
- Martin M: Treatment of congestive heart failure as a neuroendocrine disorder, *J Small Anim Pract* 44(4):154-160, 2003.
- Sisson D, Kittleson MD: Management of heart failure: principles of treatment, therapeutic strategies, and pharmacology. In Fox PR, Sisson D, Moise NS, editors: *Textbook of canine and feline cardiology*, ed 2, Philadelphia, 1999, WB Saunders.
- Ware WA, Bonagura JD: Pulmonary edema. In Fox PR, Sisson D, Moise NS, editors: *Textbook of canine and feline cardiology*, ed 2, Philadelphia, 1999, WB Saunders.

CANINE CAVAL SYNDROME OF HEARTWORM DISEASE

Caval syndrome resulting from severe heartworm disease is caused by the rapid maturation of a large quantity of adult worms in the right atrium and cranial and caudal venae cavae. Most cases of caval syndrome occur in regions of the world where heartworm disease is highly endemic and dogs spend a large portion of time living outdoors. Caval syndrome is recognized by the following clinical signs and results of biochemical analyses: acute renal and hepatic failure, enlarged right atrium and posterior vena cava, ascites, hemoglobinuria, anemia, acute collapse, respiratory distress, DIC, jugular pulses, circulating microfilariae, and sometimes tricuspid insufficiency.

Immediate action

Immediate action in cases of caval syndrome in dogs involves immediate stabilization of the cardiovascular and respiratory systems with supplemental oxygen, furosemide (4 mg/kg IV), and careful crystalloid fluid infusion.

Diagnosis

Diagnosis of caval syndrome is based on clinical signs of cardiogenic shock with right ventricular heart failure, intravascular hemolysis, and renal and hepatic failure. Thoracic radiographs reveal cardiomegaly of the right side and enlarged tortuous pulmonary arteries. A right axis deviation may be seen on ECG tracings. Clinicopathologic changes observed include azotemia, inflammatory leukogram, regenerative anemia, eosinophilia, elevated hepatocellular enzyme activities, hemoglobinuria, and proteinuria. Circulating microfilariae may be observed on peripheral blood smears or in the buffy coat of microhematocrit tubes. Heart worm antigen tests will be strongly positive. Echocardiographic changes include visualization of a large number of heartworms in the right atrium, pulmonary arteries, and vena cava, tricuspid insufficiency, and right atrial and ventricular enlargement.

Management

Treatment involves surgical removal of as many of the adult heartworms as possible from the right jugular vein and right atrium. Glucocorticosteroids are recommended to decrease inflammation and microangiopathic disease associated with heartworm infection. For more long-term management, administer adulticide therapy several weeks following surgery, followed by routine microfilaricide therapy and then prophylaxis.

Additional Reading

- Calvert CA, Rawlings CA, McCall JW: Canine heartworm disease. In Fox PR, Sisson D, Moise NS, editors: *Textbook of canine and feline cardiology*, ed 2, Philadelphia, 1999, WB Saunders.
- Hidaka Y, Hagio M, Morakami T, et al: Three dogs under 2 years of age with heartworm caval syndrome, *J Vet Med Sci* 65(10):1147-1149, 2003.
- Kitagawa H, Kitoh K, Ohba Y, et al: Comparison of laboratory results before and after surgical removal of heartworms in dogs with vena caval syndrome, *J Am Vet Med Assoc* 213(8):1134-1136, 1998.
- Kuntz CA, Smith-Carr S, Huber M, et al: Use of a modified surgical approach to the right atrium for retrieval of heartworms in a dog, *J Am Vet Med Assoc* 208(5):692-604, 1996.

PERICARDIAL EFFUSION AND PERICARDIOCENTESIS

Pericardial effusion often develops as a consequence of neoplasia in the older dog and cat. The most common types of neoplasia that affect the heart and pericardium include hemangiosarcoma, chemodectoma, mesothelioma, and metastatic neoplasia. More rarely, other causes of pericardial effusion include benign idiopathic pericardial effusion, coagulopathy, left atrial rupture in dogs with chronic mitral valvular insufficiency, infection, or pericardial cysts. Regardless of the cause of the effusion, the development of pericardial tamponade adversely affects cardiac output.

Cardiac output is a function of heart rate and stroke volume. Stroke volume depends on cardiac preload. The presence of pericardial effusion can impede venous return to the heart and thus adversely affect preload. In addition, as preload decreases, heart rate reflexively increases in an attempt to maintain normal cardiac output. As heart rate increases more than 160 beats/minute, diastolic filling is impaired further, and cardiac output further declines. Animals with pericardial effusion often demonstrate the classic signs of hypovolemic or cardiogenic shock: anorexia, weakness, lethargy, cyanosis, cool peripheral extremities, tachycardia, weak thready pulses, hypotension, and collapse. Physical examination abnormalities may include muffled heart sounds, thready femoral pulses, pulsus paradoxus, jugular venous distention, weakness, tachycardia, cyanosis, and tachypnea. Electrocardiogram findings may include low amplitude QRS complexes (<0.5 mV), sinus tachycardia, ventricular dysrhythmias, or electrical alternans (Figure 1-39). Thoracic radiographs often demonstrate a globoid cardiac silhouette, although the cardiac silhouette rarely may appear normal with concurrent clinical signs of cardiogenic shock in cases of acute hemorrhage. In such cases the removal of even small amounts of pericardial effusion by pericardiocentesis can increase cardiac output exponentially and alleviate clinical signs (Table 1-35). Unless an animal is dying before your eyes, ideally perform an echocardiogram to attempt to



Figure 1-39: An example of electrical alternans. This rhythm is observed in cases of pericardial effusion, as the heart swings to and fro within the fluid, toward and away from the electrical axis.

TABLE 1-35 Differential Diagnosis of Pericardial Effusion		
Type of pericardial effusion	Cause	Characteristic features
Hemorrhagic	Heart base tumors	Usually brachycephalic breeds; >8 years old; blood usually nonclotting
	Hemangiosarcoma	
	Metastatic neoplasia	
	Benign idiopathic pericardial effusion	
	Cardiac puncture	
Transudate	Physical trauma	Small breeds, >8 years of age; chronic valvular disease
	Left atrial rupture	
	Coagulopathy	
	Congestive heart failure	
	Hypoproteinemia	
Exudate	Following peritoneo-pericardial diaphragmatic hernia	Radiograph or echocardiogram will usually demonstrate lesion
	Infectious pericarditis	
	Suppurative pericarditis	Exudate in distemper, leptospirosis, and systemic fungal infection Foreign body or hematologic spread of inflammatory process

determine whether a right atrial, right auricular, or heart base mass is present before pericardiocentesis.

Pericardiocentesis

Before attempting pericardiocentesis, assemble all of the required supplies (Box 1-39).

To perform pericardiocentesis, follow this procedure:

1. Place the patient in sternal or lateral recumbency.
2. Attach ECG leads to monitor the patient for dysrhythmias during the procedure.
3. Clip a 6-cm square caudal to the right elbow over the fifth to seventh intercostal space.
4. Aseptically scrub the clipped area, and infuse 1 to 2 mg/kg of 2% lidocaine mixed with a small amount of sodium bicarbonate just dorsal to the sternum at the sixth intercostal space. Bury the needle to the hub, and inject the lidocaine as you withdraw the needle.

BOX 1-39 SUPPLIES REQUIRED FOR PERICARDIOCENTESIS

- 2% lidocaine
- 3-mL syringe
- 25-gauge needle
- No. 11 scalpel blade
- 14- to 16-gauge Abbott-T catheter or Turkel thoracic drainage catheter
- Intravenous extension tubing
- Three-way stopcock
- 60-mL syringe
- Red- and lavender-topped tubes
- Collection bowl
- Clippers
- Antimicrobial scrub

5. While the local anesthetic is taking effect, assemble the intravenous extension tubing, three-way stopcock, and 60-mL syringe.
6. Wearing sterile gloves, make a small nick incision in the skin to decrease drag on the needle and catheter during insertion.
7. Slowly insert the needle and catheter, watching for a flash of blood in the hub of the needle, and simultaneously watching for cardiac dysrhythmias on the ECG monitor.
8. Once a flash of blood is observed in the hub of the needle, advance the catheter off of the stylette further into the pericardial sac, and remove the stylette.
9. Attach the length of intravenous extension tubing to the catheter, and have an assistant withdraw the fluid slowly.
10. Place a small amount of fluid in a red-topped tube, and watch for clots. Clot formation could signify that you have penetrated the right ventricle inadvertently or that active hemorrhage is occurring. Withdraw as much of the fluid as possible, and then remove the catheter. Monitor the patient closely for fluid reaccumulation and recurrence of clinical signs of cardiogenic shock.

Additional Reading

- Less RD, Bright JM, Orton EC: Intrapericardial cyst causing cardiac tamponade in a cat, *J Am Anim Hosp Assoc* 36(2):115-119, 2000.
- MacGregor JM, Rozanski EA, McCarthy RJ, et al: Cholesterol-based pericardial effusion and aortic thromboembolism in a 9-year-old mixed breed dog with hypothyroidism, *J Vet Intern Med* 18(3):354-358, 2004.
- Machida N, Tanaka R, Takemura N, et al: Development of pericardial mesothelioma in a golden retriever with a long-term history of idiopathic pericardial haemorrhagic effusion, *J Comp Pathol* 131(2-3):166-175, 2004.
- Shubitz LE, Matz ME, Noon TH, et al: Constrictive pericarditis secondary to *Coccidioides immitis* infection in a dog, *J Am Vet Med Assoc* 218(4):537-540, 2001.
- Stafford Johnson M, Martin M, Binn S, et al: A retrospective study of clinical findings, treatment and outcome in 143 dogs with pericardial effusion, *J Am Anim Pract* 45(11):546-552, 2004.
- Zoia A, Hughes D, Connolly DJ: Pericardial effusion and cardiac tamponade in a cat with extra-nodal lymphoma, *J Small Anim Pract* 45(9):467-471, 2004.

EAR EMERGENCIES**FOREIGN BODIES**

Foreign bodies within the ear canal (e.g., foxtails) can present as emergencies because of acute inflammation and pressure necrosis of the tissue of the external auditory meatus causing pain and discomfort. Clinical signs may be limited to incessant head shaking or scratching of the ear canal.

Immediate action/treatment

Complete examination of the ear canal and removal of any foreign body often requires administration of a short-acting anesthetic agent. Once the animal has been restrained sufficiently and placed under anesthesia, carefully examine the ear canal and remove any foreign material with an alligator forceps. Stimulation of the ear canal can cause awakening

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and shaking of the head. Use care to not perforate the tympanum or cause trauma to the ear canal with the forceps. Heat-fix any purulent material within the ear canal and examine it cytologically for bacteria or fungal organisms. Gently irrigate the ear canal with warm sterile saline to remove excessive debris and exudates. Use care to avoid excessive pressure (>50 mm Hg) to avoid iatrogenic damage to the tympanic membrane.

Management

After removal of all debris and detritus, gently wipe the internal and external ear canal with a sterile gauze. Place a topical antimicrobial-antifungal-steroid ointment such as Otomax in the ear every 8 to 12 hours. If pain and discomfort is severe, systemically effective opioids or NSAIDs may be required.

OTITIS EXTERNA

Otitis externa is a common emergency that causes excessive head shaking, scratching, and purulent malodorous aural discharge.

Immediate action/treatment

Clean the ear canal with an irrigating solution such as Epiotic and wipe it clean of debris. Perform a complete aural examination to determine whether a foreign body or tumor is present and whether the tympanic membrane is intact. Heat-fix any discharge and examine it cytologically for bacteria and fungal organisms. Following careful cleansing, instill a topical antibiotic-antifungal-steroid ointment.

Management

In severe cases in which the ear canal has scarred and closed down with chronicity, consider administering systemically effective antibiotics (cephalexin, 22 mg/kg PO tid) and antifungal agents (ketoconazole, 10 mg/kg PO q12h) instead of topical therapy. Systemically effective steroids (prednisone or prednisolone, 0.5 mg/kg PO q12h) may be indicated in cases of severe inflammation to decrease pruritus and patient discomfort.

OTITIS INTERNA

Presentation of a patient with otitis interna often is characterized by torticollis, head tilt, nystagmus, circling to the affected side, or rolling. Fever, pain, vomiting, and severe depression may accompany clinical signs. Most cases of severe otitis interna are accompanied by severe otitis media. Both conditions must be treated simultaneously. The most common causes of otitis interna are *Staphylococcus aureus*, *Pseudomonas*, *Escherichia coli*, or *Proteus* spp. Otitis interna can develop by infection spreading across the tympanic membrane, through the eustachian tubes, or by hematogenous spread from the blood supply to the middle ear. In most cases of otitis media, the tympanic membrane is ruptured.

Immediate action

Perform a culture and susceptibility test of the debris behind the tympanic membrane and within the aural canal. Carefully clean the external ear canal. Medicate with a topical combination antibiotic, antifungal, and antibiotic ointment. Administer high-dose antibiotics (cephalexin, 22 mg/kg PO q8h, or enrofloxacin, 10 to 20 mg/kg PO q24h).

Management

If the tympanic membrane is not ruptured but appears swollen and erythematous, a myringotomy may need to be performed. If clinical signs of otitis media persist despite topical and systemic therapy, radiographic or CT/MRI examination of the tympanic bullae may be required.

AURAL HEMATOMA

Chronic shaking of the head and ears or aural trauma (bite wounds) causes disruption of the blood vessels and leads to the development of unilateral or bilateral aural hematomas.

Aural hematomas are clinically significant because they cause patient discomfort and are often due to the presence of some other underlying problem such as otitis externa, atopy, or aural foreign bodies. Acute swelling of the external ear pinna with fluid is characteristic of an aural hematoma. In some cases, swelling can be so severe that the hematoma breaks open, bathing the patient and external living environment in blood.

Immediate action

When a patient has an aural hematoma, investigate the underlying cause. Perform a complete aural examination to determine whether an aural foreign body, otitis externa, or atopy are present. Carefully examine and gently clean the inner ear canal. Treat underlying causes.

Management

Management of an aural hematoma involves draining the hemorrhagic fluid from the aural tissue and tacking the skin down in multiple places to prevent reaccumulation of fluid until the secondary cause is resolved. Many techniques have been described to surgically tack down the skin overlying the hematoma. After the animal has been placed under general anesthesia, lance the hematoma down the middle with a scalpel blade and remove the fluid and blood clot. Tack down the skin with multiple through-and-through interrupted or mattress sutures through the ear. Some clinicians prefer to suture through and attach a sponge or length of x-ray film to the front and back of the ear for stabilization and support. More recently, a laser can be used to drill holes in the hematoma and tack the skin down in multiple areas. Compress the ear against the head with a compression bandage, whenever possible, for 5 to 7 days after the initial surgery, and then recheck the ear. The patient must wear an Elizabethan collar until the surgical wound and hematoma heal to prevent self-mutilation. Also systemically treat underlying causative factors such as otitis externa with antibiotics, antifungals, and steroids as indicated. Investigate and treat other underlying causes such as hypothyroidism or allergies.

Additional Reading

- Bass M: Symposium on otitis externa in dogs, *Vet Med* 99(3):252, 2004.
- Dye TL, Teague HD, Ostwald DO, et al: Evaluation of a technique using the carbon dioxide laser for the treatment of aural hematomas, *J Am Anim Hosp Assoc* 38(4):385-390, 2002.
- Gothelf LN: Diagnosis and treatment of otitis media in dogs and cats, *Vet Clin North Am Small Anim* 34(2):469-487, 2004.
- Lanz OI, Wood BC: Surgery of the ear and pinna, *Vet Clin North Am Small Anim Pract* 34(2): 567-599, 2004.
- Murphy KM: A review of the techniques for the investigation of otitis externa and otitis media in dogs and cats, *Clin Tech Small Anim Pract* 16(4):236-241, 2001.

ELECTROCUTION/ELECTRIC SHOCK

Electrocution usually is observed in young animals after they have chewed on an electric cord. Other causes of electrocution include use of defective electrical equipment or being struck by lightning. Electric current passing through the body can produce severe dysrhythmias, including supraventricular or ventricular tachycardia and first- and third-degree atrioventricular block. The electric current also can produce tissue destruction from heat and electrothermal burns. Electrocution also commonly results in noncardiogenic pulmonary edema caused by massive catecholamine release and increase in pulmonary vascular pressures during the event. Ventricular fibrillation can occur, although that depends on the intensity and path of the electrical current and duration of contact.

Clinical signs of electrocution include acute onset of respiratory distress with moist rales, and localized necrosis or thermal burns of the lips and tongue. Often the skin at the commissures of the mouth appears white or yellow and firm to the touch. Muscle fasciculations, loss of consciousness, and ventricular fibrillation may occur. Thoracic radiographs often reveal an increased interstitial to alveolar lung pattern in the dorsocaudal lung fields.

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Noncardiogenic pulmonary edema can develop up to 24 to 36 hours after the initial incident. The first 24 hours are most critical for the patient, and then prognosis improves.

The most important aspect in the treatment of the patient with noncardiogenic pulmonary edema is to minimize stress and to provide supplemental oxygen, with positive pressure ventilation, when necessary. Although treatment with vasodilators (low-dose morphine) and diuretics (furosemide) can be attempted, noncardiogenic pulmonary edema is typically resistant to vasodilator and diuretic therapy. Positive inotropes and pressor drugs may be necessary to treat shock and hypotension. Opioid drugs (morphine, hydromorphone, oxycodone) may be useful in controlling anxiety until the pulmonary edema resolves. Administer broad-spectrum antibiotics (cefazolin; amoxicillin and clavulanic acid [Clavamox]) to treat thermal burns. Use analgesic drugs to control patient discomfort. If thermal burns are extensive and prohibit adequate food intake, place a feeding tube as soon as the patient's cardiovascular and respiratory function are stable and the patient can tolerate anesthesia.

EMERGENCIES OF THE FEMALE REPRODUCTIVE TRACT AND GENITALIA

UTERINE PROLAPSE

Prolapse of the uterus occurs in the immediate postparturient period in the bitch and queen. Excessive straining during or after parturition causes the uterus to prolapse caudally through the vagina and vulva. Immediate intervention is necessary. Examine the bitch or queen for a retained fetus. Treatment consists of general anesthesia to replace the prolapsed tissue. If the uterus is edematous, physical replacement may be difficult or impossible. Application of a hypertonic solution such as hypertonic (7%) saline or dextrose (50%) to the exposed endometrium can help shrink the tissue. That, combined with gentle massage to stimulate uterine contraction and involution and lubrication with sterile lubricating jelly, can aid in replacement of the organ into its proper place. To ensure proper placement in the abdominal cavity and to prevent recurrence, perform an exploratory laparotomy and hysteropexy. Postoperatively, administer oxytocin (5 to 20 units IM) to cause uterine contraction. If the uterus contracts, it is usually not necessary to suture the vulva. Administer antibiotics postoperatively. Recurrence is uncommon, even with subsequent pregnancies.

If the tissue is damaged or too edematous to replace or if the tissue is devitalized, traumatized or necrotic, perform an ovariohysterectomy. In some instances, replacement of the damaged tissue is not necessary before removal.

PYOMETRA

Pyometra occurs in dogs and cats. The disease process occurs as a result of infection overlying cystic endometrial hyperplasia under the constant influence of progesterone. During the 2-month luteal phase after estrus or following copulation, artificial insemination, or administration of hormones (particularly estradiol or progesterone), the myometrium becomes relaxed and favors a quiescent environment for bacterial proliferation.

Clinical signs of pyometra are associated with the presence of bacterial endotoxin and sepsis. Early, affected animals become lethargic and anorectic. Polyuria with secondary polydipsia is often present because of the influence of bacterial endotoxin on renal tubular concentration. If the cervix is open, purulent or mucoid vaginal discharge may be observed. Later in the course of pyometra, vomiting, diarrhea, and progressive debilitation resulting from sepsis occur. Diagnosis is based on clinical signs in an intact queen or bitch and radiographic or ultrasonographic evidence of a fluid-filled tubular density in the ventrocaudal abdomen, adjacent to the urinary bladder (Figures 1-40 and 1-41).

Treatment of open and closed pyometra is correction of fluid and electrolyte abnormalities, administration of broad-spectrum antibiotics, and ovariohysterectomy. Closed pyometra is a life-threatening septic condition. Open pyometra also can become life-threatening and so should be treated aggressively. In closed pyometra, conservative medical therapy is not advised. Administration of prostaglandins and oxytocin do not reliably cause the cervix



Figure 1-40: An example of a large pus-filled uterus after emergency ovariohysterectomy.

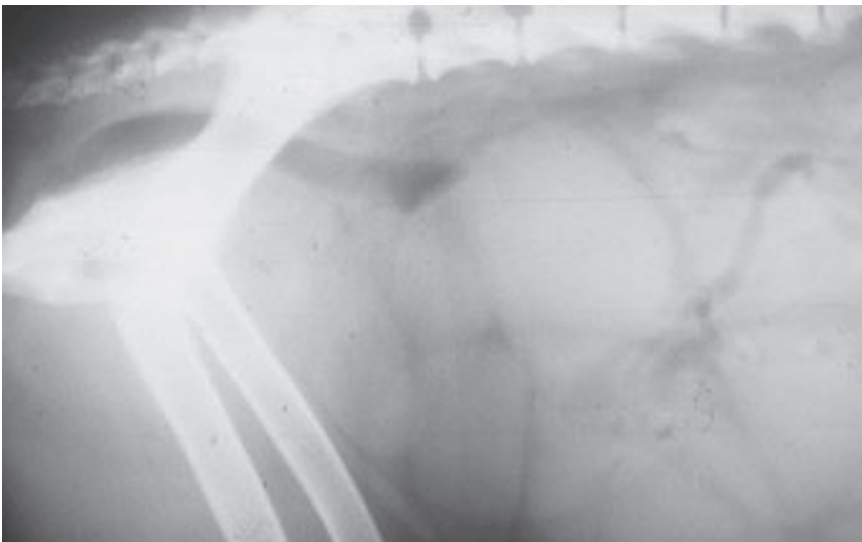


Figure 1-41: Abdominal radiograph of a pyometra. Note the fluid-filled soft tissue density in the caudal abdomen.

to open and can result in ascending infection from the uterus into the abdomen or uterine rupture, both of which can result in severe peritonitis.

For animals with an open pyometra, ovariohysterectomy is the most reliable treatment for chronic cystic endometrial hyperplasia. Although less successful than ovariohysterectomy, medical therapy may be attempted in breeding bitches as an alternative to surgery. The most widely used medical therapy in the breeding queen and bitch is administration of prostaglandin $F_{2\alpha}$. This drug has not been approved for use in the queen or bitch in the United States. To proceed with medical management of pyometra, first determine the size

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of the uterus. Start the patient on antibiotic therapy (ampicillin, 22 mg/kg IV q6h, or enrofloxacin, 10 mg/kg PO q24h). Administer the prostaglandin $F_{2\alpha}$ (250 μ g/kg SQ q24h) for 2 to 7 days until the size of the uterus approaches normal. Measure serum progesterone concentrations if the bitch is in diestrus. As the corpus luteum degrades under the influence of prostaglandin $F_{2\alpha}$, serum progesterone levels will decline.

Prostaglandin $F_{2\alpha}$ is an abortifacient and thus should not be administered to the pregnant bitch or queen. Clinical signs of a reaction to prostaglandin $F_{2\alpha}$ can occur within 5 to 60 minutes in the bitch and can last for as long as 20 minutes. Clinical signs of a reaction include restlessness, hypersalivation, panting, vomiting, defecation, abdominal pain, fever, and vocalization. In a very ill animal, death can occur. The efficacy of prostaglandin $F_{2\alpha}$ is limited and may require more than one treatment. The bitch should be bred on the next heat cycle and then spayed because progressive cystic endometrial hyperplasia will continue to occur.

ACUTE METRITIS

Acute metritis is an acute bacterial infection of the uterus that typically occurs within 1 to 2 weeks after parturition. The most common organism observed in metritis is *E. coli* ascending from the vulva and vaginal vault. Sepsis can progress rapidly. Clinical signs of acute metritis include inability to nurse puppies, anorexia, lethargy, foul-smelling purulent-sanguineous vaginal discharge, vomiting, or acute collapse.

Physical examination may reveal fever, dehydration, and a turgid distended uterus. Septic inflammation will be observed on vaginal cytologic examination. An enlarged uterus can be observed with abdominal radiographs and ultrasonography.

Treatment of acute metritis is directed at restoring hydration status with intravenous fluids and treating the infection with antibiotics. Because the primary cause of metritis is *E. coli* infection, start enrofloxacin (10 mg/kg IV or PO once daily) therapy. As soon as the patient's cardiovascular status is stable enough for anesthesia, perform an ovariohysterectomy. If the patient is not critical and is a valuable breeding bitch, medical therapy can be attempted. Medical management of acute bacterial metritis includes administration of oxytocin (5 to 10 units q3h for three treatments) or administration of prostaglandin $F_{2\alpha}$ (250 μ g/kg/day for 2 to 5 days) to evacuate the uterine exudate and increase uterine blood flow. Either drug should be used concurrently with antibiotics.

UTERINE RUPTURE

Rupture of the gravid uterus is rare in cats and dogs but has been reported. Uterine rupture may occur as a consequence of parturition or result from blunt abdominal trauma. Feti expelled into the abdominal cavity may be resorbed but more commonly cause the development of peritonitis. If fetal circulation is not disrupted, the fetus actually may live to term. Uterine rupture is an acute surgical emergency. An ovariohysterectomy with removal of the extrauterine puppies and membranes is recommended. If only one horn of the uterus is affected, a unilateral ovariohysterectomy can be performed to salvage the remaining unaffected puppies and preserve the breeding potential for the valuable bitch. If uterine rupture occurs because of pyometra, peritonitis is likely, and copious peritoneal lavage should be performed at the time of surgery. The patient should be placed on 7 to 14 days of antibiotic therapy (amoxicillin or amoxicillin and clavulanic acid [Clavamox] with enrofloxacin).

VAGINAL PROLAPSE

Vaginal prolapse occurs from excessive proliferation and hyperplasia of vaginal tissue while under the influence of estrogen during proestrus (Figure 1-42). The hyperplastic tissue usually recedes during diestrus but reappears with subsequent heat cycles. Vaginal prolapse can be confused with vaginal neoplasia. The former condition occurs primarily in younger animals, whereas the latter condition occurs primarily in older animals. Treatment for vaginal hyperplasia or prolapse generally is not required if the tissue remains within the vagina. The proliferation can lead to dysuria or anuria, however. In some cases, the tissue becomes



Figure 1-42: Vaginal prolapse in a bitch.

dried out and devitalized or becomes traumatized by the animal. Such extreme cases warrant immediate surgical intervention. The treatment for vaginal prolapse consists of ovariectomy to remove the influence of estrogen, placement of an indwelling urinary catheter if the patient is dysuric, and protection of the hyperplastic tissue until it recedes on its own. Although surgical resection of the hyperplastic tissue has been recommended, excessive hemorrhage after removal can occur, and so the procedure should not be attempted. The patient should wear an Elizabethan collar at all times to prevent self-mutilation. Administer broad-spectrum antibiotics for a minimum of 7 to 14 days or until the hyperplastic tissue recedes. Keep the tissue clean with saline solution.

EMERGENCIES OF PREGNANCY AND PARTURITION

Dystocia

Dystocia, or difficult birth, can occur in the dog and cat but is more common in the dog. A diagnosis of dystocia is made based on the time of onset of visible labor and the time in which the last puppy or no puppy has been born, the intensity and timing of contractions, the timing of when the amniotic membranes first appear, the condition of the bitch, and the timing of gestation. Causes of dystocia can be maternal or fetal and include primary or secondary uterine inertia, narrowing of the pelvic canal, hypocalcemia, psychological disturbances, or uterine torsion. Maternal-fetal disproportion, or large fetus size in relation to the bitch or queen, also can result in dystocia (Box 1-40).

Obtain an abdominal radiograph for all cases of suspected dystocia at the time of presentation to determine the size of the fetus, presentation of the fetus (Both anterior or posterior presentation can be normal in the bitch or queen, but fetal malpositioning can

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BOX 1-40 DIAGNOSTIC CRITERIA FOR DYSTOCIA

- Fetus lodged in birth canal
- Presence of vaginal stricture or band of tissue preventing normal delivery
- Prolonged gestation (>70 days)
- Drop in rectal temperature (<100° F) with no evidence of labor
- Green vaginal discharge and no evidence of delivery of fetus
- No puppies delivered after 2 to 3 hours of a visible amniotic sac
- Strong contractions with no puppy or kitten delivered after 30 minutes
- Weak, infrequent contractions with no puppy delivered in 4 hours from onset of labor
- More than 2 hours have passed with no evidence of further contraction or delivery of a puppy
- Signs of systemic illness or pain: depression, weakness, sepsis

cause dystocia), and whether there is radiographic evidence of a uterine rupture or torsion. If maternal-fetal disproportion, uterine torsion, or uterine rupture is observed, take the patient immediately to surgery. If the puppies or kittens are in a normal position for birth, medical management can be attempted.

Clip the perineum and aseptically scrub it. Wearing sterile gloves, insert a lubricated finger into the vagina and palpate the cervix. Massage (or “feather”) the dorsal wall of the vagina to stimulate contractions. Place an intravenous catheter, and administer oxytocin (2 to 20 units IM), repeating up to 3 times at 30-minute intervals. In some cases, hypoglycemia or hypocalcemia can contribute to uterine inertia. Administration of a calcium-containing solution (lactated Ringer’s solution) with 2.5% dextrose is advised. Alternately, administer 10% calcium gluconate (100 mg/5 kg IV slowly). If labor has not progressed after 1 hour, immediately perform a cesarean section.

Uterine torsion

Uterine torsion is an uncommon emergency seen in the gravid and nongravid uterus and has been reported in dogs and cats. The onset of clinical signs of abdominal pain and straining as if to whelp/queen or defecate is usually acute and constitutes a surgical emergency. In some cases, there may have been a history of delivery of a live or dead fetus. Vaginal discharge may or may not be present. Radiographs or ultrasound examination reveal a fluid-filled or air-filled tubular density in the ventral abdomen. Treatment consists of placing an intravenous catheter, stabilizing the patient’s cardiovascular status with intravenous fluids and sometimes blood products, and performing an immediate ovariohysterectomy. If there are viable feti, the uterus should be delivered *en mass* and the puppies or kittens delivered.

Spontaneous abortion

The expulsion of one or more fetus before term is known as spontaneous abortion. In dogs and cats, it is possible to expel or abort one or more fetuses and still carry viable fetuses to term and deliver normally. Clinical signs of spontaneous abortion include vaginal discharge and abdominal contractions. In some cases, the fetus is found, or there may be evidence of fetal membranes or remnants. Causes of spontaneous abortion in dogs include *Brucella canis*, herpesvirus, coronavirus, and toxoplasmosis. In cats, herpesvirus, coronavirus, and feline leukemia virus can cause spontaneous abortion. In both species, trauma, hormonal factors, environmental pathogens, drugs, and fetal factors also can result in spontaneous abortion.

Pregnancy termination in the bitch and queen

The safest method of pregnancy termination in the bitch or queen is by performing an ovariohysterectomy. Oral diethylstilbesterol is not an effective mechanism of pregnancy termination in the bitch. A so-called mismating shot, an injection of estradiol cypionate (0.02 mg/lb IM) is effective at causing termination of an early pregnancy but can be

associated with severe side effects, including bone marrow suppression and pyometra. Estradiol cypionate is not approved for use in the bitch or queen and is not recommended.

Prostaglandin $F_{2\alpha}$ is a natural abortifacient in the bitch if treatment is started within 5 days of cytologic evidence of diestrus (noncornified epithelium on a vaginal smear). The prostaglandin $F_{2\alpha}$ causes lysis of the corpora lutea and a rapid decline in progesterone concentration. The prostaglandin $F_{2\alpha}$ is administered for a total of eight injections (250 $\mu\text{g}/\text{kg}$ q12h for 4 days), along with atropine (100 to 500 $\mu\text{g}/\text{kg}$ SQ). Side effects can occur within 5 to 40 minutes of injection and include restlessness, panting, salivation, abdominal pain, urination, vomiting, and diarrhea. Walking the patient for 20 to 30 minutes after each treatment sometimes decreases the intensity of the reactions.

Bitches in the first half of the pregnancy often resorb the embryos. If prostaglandin $F_{2\alpha}$ is administered in the second half of the pregnancy, the fetuses are aborted within 5 to 7 days of treatment. Measure serum progesterone concentrations at the end of treatment to ensure complete lysis of the corpus luteum. Prostaglandin $F_{2\alpha}$ is not approved for pregnancy termination in the bitch.

In cats, prostaglandin $F_{2\alpha}$ can terminate pregnancy after day 4 of gestation. Prostaglandin $F_{2\alpha}$ should be used only in healthy queens (100 to 250 $\mu\text{g}/\text{kg}$ SQ q24h for 2 days). Side effects in the queen are similar to those observed in the bitch but typically have a shorter duration (2 to 20 minutes). Prostaglandin $F_{2\alpha}$ is not approved for use in cats in the United States. The use of prostaglandin $F_{2\alpha}$ does not preclude breeding and pregnancy at a later date.

Additional Reading

- Biddle D, Macintire DK: Obstetrical emergencies, *Clin Tech Small Anim Pract* 15(2):88-93, 2000.
- Drobatz KJ, Mandell DC, Neath P: Urinary bladder herniation thru a vaginal tear in a Rottweiler with dystocia, *J Vet Emerg Crit Care* 10(3):173-175, 2000.
- Greenberg D, Yates D: What is your diagnosis? Vaginal hyperplasia, *J Small Anim Pract* 43(9):381, 406, 2002.
- Hayes G: Asymptomatic uterine rupture in a bitch, *Vet Rec* 154(14):438-439, 2004.
- Jutkowitz LA: Reproductive emergencies, *Vet Clin North Am Small Anim* 35:397-420, 2005.
- Lucas X, Agut A, Ramis G, et al: Uterine rupture in a cat, *Vet Rec* 152(10):301-302, 2003.
- Misumi K, Fujiki M, Miura N, et al: Uterine torsion in two non-gravid bitches, *J Small Anim Pract* 41(10):468-471, 2000.
- Ridyard AE, Welsh EA, Gunn-Moore DA: Successful treatment of uterine torsion in a cat with severe metabolic and haemostatic complications, *J Feline Med Surg* 2(2):115-119, 2000.

EMERGENCIES OF THE MALE GENITALIA AND REPRODUCTIVE TRACT

Figure 1-43 illustrates conditions of the male genitalia and reproductive tract that require emergent care.

SCROTAL TRAUMA

In the dog and cat the majority of injuries to the scrotum are associated with animal fights or shearing and abrasive injuries sustained in accidents involving automobiles. Scrotal injuries should be categorized as superficial or penetrating.

Treatment of superficial injuries to the scrotum includes cleaning the wound with dilute antimicrobial cleanser and drying it. Administer antiinflammatory doses of steroids (prednisolone, 0.5 to 1.0 mg/kg PO q12-24h) or NSAIDs (carprofen, 2.2 mg/kg PO q12h in dogs) for the first several days after scrotal injury to prevent or treat edema. Administer topical antibiotic ointment until the wound heals. In most cases, place an Elizabethan collar to prevent self-mutilation. Prognosis is generally favorable; however, semen quality may be affected for months after injury because of scrotal swelling and increased scrotal temperature.

Penetrating injuries to the scrotum are more serious and are associated with severe swelling and infection. Surgically explore and debride penetrating scrotal wounds. Administer systemically effective antibiotics and analgesics. In extreme cases, particularly those that involve the testicle, consider castration and scrotal ablation.

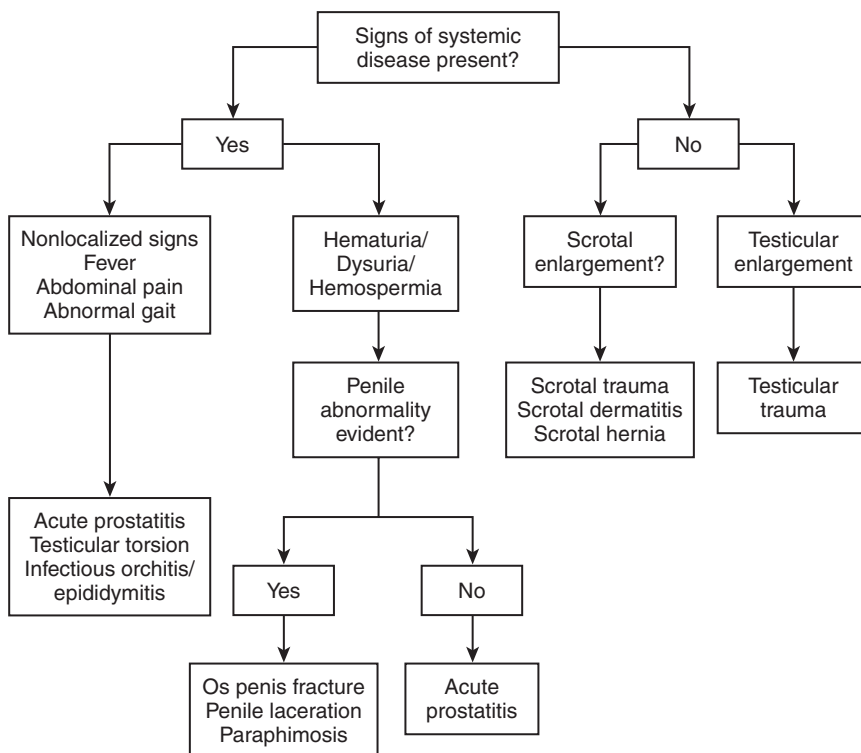


Figure 1-43: Emergencies of the male genitalia and reproductive tract.

ACUTE SCROTAL DERMATITIS

Scrotal dermatitis is common in intact male dogs and can be associated with direct physical injury, self-infliction from licking, chemical irritation, burns, or contact dermatitis. In affected animals, the scrotum can become extremely inflamed, swollen, and painful. If left untreated, pyogranulomatous dermatitis can develop.

Make an attempt to determine whether an underlying systemic illness is present that could predispose the animal to scrotal dermatitis. Widespread vasculitis with scrotal edema, pain, fever, and dermatitis has been associated with *Rickettsia rickettsii* (Rocky Mountain spotted fever) infection. *Brucella canis* also has been associated with scrotal irritation and dermatitis. If scrotal dermatitis follows from an infectious cause, empiric use of glucocorticosteroids potentially can make the condition worse by suppressing immune function. Empiric treatment with antibiotics also potentially can confound making an accurate diagnosis.

Treatment of scrotal dermatitis is to eliminate predisposing causes, if possible. Place an Elizabethan collar at all times to prevent self-mutilation. Bathe the scrotum with a mild antimicrobial soap and dry it to remove any offending chemical irritants. Topical medications including tar shampoo, tetracaine, neomycin, and petroleum can cause further irritation and are contraindicated. Use oral or parenteral administration of glucocorticosteroids or NSAIDs to control discomfort and inflammation.

SCROTAL HERNIA

Scrotal hernias occur when the contents of the abdomen (intestines, fat, mesentery, omentum) protrude through the inguinal ring into the scrotal sac. Like inguinal hernias, scrotal

hernias are surgical emergencies only if intestinal incarceration or vascular obstruction occurs. Differential diagnoses for scrotal hernias include epididymitis, orchitis, testicular torsion, and testicular neoplasia.

Definitive therapy for a scrotal hernia involves exploratory laparotomy and surgical reduction of the contents of the hernia, surgical correction of the rent in the inguinal ring, and castration.

TESTICULAR TRAUMA

Trauma to the epididymis or testicle can cause testicular pain and swelling of one or both testes. Treat penetrating trauma to the testicle by castration to prevent infection and self-mutilation. Administer oral antibiotics (amoxicillin or amoxicillin-clavulanate) for 7 to 10 days after the injury. Nonpenetrating injuries to the scrotum and testicle rarely may cause acute testicular hemorrhage or hydrocele formation. Palpation of the affected area often reveals a peritesticular, soft, compliant area. Treatment consists of cool compresses on the scrotum and testicle and administration of antiinflammatory doses of glucocorticosteroids or NSAIDs. If the swelling does not resolve spontaneously in 5 to 7 days, consider surgical exploration and drainage. Increased scrotal temperature and testicular inflammation can affect semen quality for months after the initial incident.

TESTICULAR TORSION

Testicular torsion, or torsion of the spermatic cord, causes rotation of the testicle, ultimately causing obstruction to venous drainage. Testicular torsion often is associated with a neoplastic mass of a retained testicle within the abdomen but also can be observed with nonneoplastic testes located within the scrotum. The predominant clinical signs are pain, stiff stilted gait, and the presence of an abnormally swollen testicle (if located within the scrotum). If an intraabdominal testicular torsion is present, pain, lethargy, anorexia, and vomiting can occur (see Acute Condition in the Abdomen). An intraabdominal mass may be palpable. Perform an abdominal or testicular ultrasound, preferably with color flow Doppler to evaluate perfusion to the testicle. Treatment involves surgical removal of the involved testes.

INFECTIOUS ORCHITIS AND EPIDIDYMITIS

Bacterial infections of the testicle or epididymis most commonly are caused by ascending infections of the normal bacterial flora of the prepuce or urethra. Common inhabitants include *Escherichia coli*, *Staphylococcus aureus*, *Streptococcus* spp., and *Mycobacterium canis*. *Brucella canis* and *R. rickettsii* are also capable of causing orchitis and epididymitis in the dog. Clinical signs of orchitis or epididymitis include testicular enlargement, stiff stilted gait, and reluctance to walk. Physical examination often reveals a fever and self-induced trauma to the scrotum from licking or chewing at the inflamed area. Collect a semen sample by ejaculation, and culture it to identify the causative organism. Alternately, collect samples by needle aspiration of the affected organ(s) and test serologically for *B. canis*.

Treatment of infectious orchitis involves a minimum of 3 to 4 weeks of specific antimicrobial therapy, based on culture and susceptibility testing, whenever possible. If a bacterial culture cannot be obtained, initiate fluoroquinolone therapy (enrofloxacin, 10 mg/kg PO q24h). Doxycycline (5 mg/kg PO bid for 7 days) has been shown to suppress but not eradicate *B. canis* infection. Testicular inflammation and increased temperature can affect sperm quality for months after infection.

ACUTE PROSTATITIS

The most common causes of acute prostatitis are associated with acute bacterial infection (*E. coli*, *Proteus* spp., *Pseudomonas* spp., and *Mycoplasma* spp.). Less common causes include fungal infection (*Blastomyces dermatitidis*) or anaerobic bacterial infection.

Acute prostatitis is characterized by fever, caudal abdominal pain, lethargy, anorexia, blood in the ejaculate, hematuria, dyschezia, and occasionally stranguria or dysuria. The patient often appears painful and depressed and may be dehydrated on physical examination.

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Symmetric or asymmetric prostatomegaly and prostate pain may be evident on rectal palpation. In severely affected dogs, clinical signs of tachycardia, hyperemic or injected mucous membranes, bounding pulses, lethargy, dehydration, and fever may be present because of sepsis. Death can occur within 2 days if a prostatic abscess ruptures.

Diagnosis of acute prostatitis is confirmed based on the presenting clinical signs, neutrophilic leukocytosis (with or without a left shift), and positive urine culture results. Prostatic samples may be obtained from the prostatic portion of the ejaculate, prostatic massage, urethral discharge, urine, or (less commonly) prostatic aspirate. Although semen samples can yield positive bacterial cultures, dogs with acute prostatitis are often unwilling to ejaculate. Radiography may reveal an enlarged prostate, but this alone does not confirm the diagnosis of prostatitis. An abdominal ultrasound often reveals prostatic abscessation and allows for the collection of samples from the affected area(s) via prostatic aspirate. Aspiration of the affected tissue potentially can wick infection into periprostatic tracks. Cytologic examination of the patient's ejaculate or prostatic wash from a dog with acute prostatitis reveals numerous inflammatory cells and may contain bacterial organisms.

The treatment of a patient with acute prostatitis is directed at correcting dysuria and constipation associated with prostatic enlargement. Enrofloxacin (10 mg/kg PO sid) can penetrate the inflamed prostatic tissue and is effective in treating gram-negative and *Mycoplasma* spp. infections. Ciprofloxacin does not appear to penetrate prostatic tissue as readily. Alternatives to enrofloxacin therapy are trimethoprim-sulfamethoxazole (30 mg/kg PO q12h) or chloramphenicol (25-50 mg/kg PO q8h) for a minimum of 2 to 3 weeks. Castration is recommended because benign prostatic hyperplasia may be a predisposing factor in the development of acute prostatitis. Do not perform castration until the patient has been on antibiotic therapy for a minimum of 7 days, to prevent the surgical complication of schirrous cords. Finasteride (Proscar, 1 mg/kg PO q24h), an antiandrogen 5 α -reductase inhibitor, may help reduce the size of prostatic tissue until the effects of castration are observed. If a prostatic abscess is present, perform marsupialization, surgical drainage, or ultrasonographic drainage. Surgical therapy is associated with a large incidence of complications, including incontinence, chronic drainage from fistulas and stomas, septic shock, and death.

Os Penis Fracture

Fracture of the os penis is an uncommon condition encountered in male dogs. Os penis fractures can occur with minimal soft tissue damage but cause hematuria and dysuria. On physical examination, urethral obstruction and crepitus in the penis are found. A lateral abdominal radiograph is usually sufficient to document the fracture. Treatment consists of conservative therapy, in most cases, and consists primarily of analgesia administration. If the urethra also is damaged, place a urethral catheter for 5 to 7 days to allow the urethral mucosa to heal. Fractures of the os penis that are comminuted or severe enough to cause urethral obstruction require open reduction and fixation, partial penile amputation, or antescrotal (prescrotal) urethrostomy.

Laceration

Lacerations of the penis cause significant bleeding because of the extensive vascular supply to the penis. Dogs and cats tend to lick penile lacerations and prevent adequate clot formation. Sedation or general anesthesia often is required to evaluate and treat the laceration. After sedation or general anesthesia, place a urinary catheter and examine the penis under a stream of cold water. Small lacerations can be managed with cold compresses and one to several absorbable sutures. Extensive suturing usually is not required. Prevent erection by isolating the patient from females in estrus or allowing excitement or excessive activity. Place an Elizabethan collar to prevent self-mutilation. Initiate systemic antibiotic therapy to prevent infection.

Paraphimosis

The inability to withdraw the penis into the prepuce in male dogs or cats is known as paraphimosis. Paraphimosis usually develops following an erection in young male dogs and in

older dogs after coitus. Mucosal edema, hemorrhage, self-mutilation, and necrosis requiring penile amputation can occur if left untreated. Treatment consists of applying cold water to the penis and reducing edema with application of an osmotic substance such as sugar. Examine the base of the penis for hair rings that can prevent retraction of the penis into the prepuce. Rinse the penis carefully with cold water and lubricate it with sterile lubricant and replace it into the prepuce. If the penis cannot be reduced easily into the prepuce, anesthetize the patient and make a small incision at the lateral aspect of the preputial opening. Replace the penis and close the incision with absorbable suture. Place a purse-string suture and leave it in place for several days to prevent recurrence. Instill topical antimicrobial ointment with steroids into the prepuce several times a day. In severe cases, a urinary catheter may need to be placed to prevent urethral obstruction, until penile swelling and edema resolve. Place an Elizabethan collar to prevent excessive licking during the healing process.

URETHRAL PROLAPSE

Prolapse of the distal urethra is a condition usually confined to intact male English Bulldogs, although isolated incidences also have been reported in Yorkshire and Boston Terriers. The exact cause of this condition is unknown but usually is associated with a condition that causes increased intraabdominal pressure or urethral straining, including sexual excitement, coughing, vomiting, obstructed airway or brachycephalic airway syndrome, urethral calculi, genitourinary tract infection, and masturbation.

The urethral prolapse usually appears as a mushroom-tip congested, irritated mass at the end of the penis that may or may not bleed (Figure 1-44). In some cases, bleeding occurs or worsens with sexual excitement. Clinical signs associated with the prolapsed



Figure 1-44: Example of urethral prolapse. This condition is most commonly observed in intact male Bulldogs, although it has been associated with neoplasia and urethral calculi in other breeds.

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urethra include excessive licking of the prepuce, stranguria, and preputial bleeding. Once the mass is observed, other differential diagnoses include transmissible venereal tumor, urethral polyp, trauma, urethritis, and neoplasia. In most cases, however, the prolapse occurs in intact young dogs, making neoplastic conditions less likely.

Treatment for urethral prolapse should occur at the time of diagnosis to prevent self-induced trauma and infection. Immediate therapy includes manual reduction of the prolapsed tissue and placement of a purse-string suture around an indwelling urinary catheter. The purse-string suture can remain in place for up to 5 days until definitive repair. Until the time of surgery, place an Elizabethan collar on the patient to prevent self-mutilation. Several forms of surgical correction have been described. In some cases, surgical resection of the prolapsed tissue with apposition of the urethral and penile mucosa can be attempted. More recently, a technique involving placement of several mattress sutures to reduce and secure the prolapsed tissue has been described. Recurrence of prolapse can occur with either technique, particularly if the inciting event recurs. Because there may be a genetic predisposition in this breed and because the prolapse can recur with sexual excitement, neutering should strongly be recommended.

Additional Reading

- Boland LE, Hardie RJ, Gregory SP, et al: Ultrasound-guided percutaneous drainage as the primary treatment for prostatic abscesses and cysts in dogs, *J Am Anim Hosp Assoc* 39(2): 151-159, 2003.
- Gobello C, Corrada Y: Noninfectious prostatic diseases in dogs, *Compend Contin Educ Pract Vet* 24(2):99-107, 2002.
- Hecht S, King R, Tidwell AS, et al: Ultrasound diagnosis: intraabdominal torsion of a non-neoplastic testicle in a cryptorchid dog, *Vet Radiol Ultrasound* 45(1):58-61, 2004.
- Kirsch JA, Hauptman JG, Walshaw R: A urethropexy technique for surgical treatment of urethral prolapse in the male dog, *J Am Anim Hosp Assoc* 38:381-384, 2002.
- Kutzler MA, Yeager A: Prostatic diseases. In Ettinger S, Feldman EC, editors: *Textbook of veterinary internal medicine*, ed 6, Philadelphia, 2005, WB Saunders.
- L'Abée-Lung TM, Heiene R, Friis NF, et al: *Mycoplasma canis* and urogenital disease in dogs in Norway, *Vet Rec* 153(8):231-235, 2003.
- Ober CP, Spaulding K, Breitschwerdt EB, et al: Orchitis in two dogs with Rocky Mountain spotted fever, *Vet Radiol Ultrasound* 45(5):458-465, 2004.

ENVIRONMENTAL AND HOUSEHOLD EMERGENCIES

FROSTBITE

Local freezing or frostbite most commonly affects the peripheral tissues of the ears, tail, paws, and genitalia that are sparsely covered with fur, are poorly vascularized, and may have been traumatized previously by cold. Clinical signs of frostbite are paleness and appearance of a blanched pink to white discoloration to the skin. The skin also may appear black and necrotic.

Immediate action

Immediate treatment consists of slowly rewarming the affected area with moist heat at 29.5° C (85° F) or by immersion in warm water baths. Analgesics may be required to alleviate patient discomfort. Carefully dry the injured areas and protect them from further trauma.

Management

The use of prophylactic antibiotics is controversial because it can promote resistant bacterial infection. Use of antibiotics should be based on the presence of infection. Treatments that are ineffective and may be harmful include rubbing the affected areas, pressure bandages, and ointments. Corticosteroids can decrease cellular immunity and promote infection and are therefore contraindicated. Many frostbitten areas that appear nonviable can regain function gradually. Use care when removing areas of necrotic tissue. Affected areas

may take several days to a week before fully manifesting areas of demarcation between healthy viable and necrotic nonviable tissue.

HYPOTHERMIA

Chilling of the entire body from exposure or immersion in extremely cold water results in a decrease in core body temperature and physiologic processes that become irreversible when the body temperature falls below 24° C (75° F). Mild hypothermia can be 32° to 37° C, moderate hypothermia from 28° to 32° C, and severe hypothermia below 28° C. The duration of exposure and the general condition of the animal influences its ability to survive.

Clinical signs and consequences associated with hypothermia include shivering, vasoconstriction, mental depression, hypotension, sinus bradycardia, hypoventilation with decreased respiratory rate, increased blood viscosity, muscle stiffness, atrial and ventricular irritability, decreased level of consciousness, decreased oxygen consumption, metabolic (lactic) acidosis, respiratory acidosis, and coagulopathies including DIC.

Immediate action

If the animal is breathing, administer warm, humidified oxygen at 4 to 10 breaths per minute. If the animal is not breathing or is severely hypoventilating, endotracheal intubation with mechanical ventilation may be necessary. Place an intravenous catheter and infuse warmed crystalloid fluids. If the blood glucose is less than 60 mg/dL, add supplemental dextrose (2.5%) to the crystalloid fluids. Monitor the core body temperature and ECG closely. Rewarming should occur in the form of external circulating warm water blankets, radiant heat, and circulating warm air blankets (Bair Hugger). *Never* use a heating pad, to avoid iatrogenic thermal burn injury. Severe hypothermia may require core rewarming in the form of intraperitoneal fluids (10 to 20 mL/kg of lactated Ringer's solution warmed to 39.4° C [103° F]). Place a temporary peritoneal dialysis catheter, and repeat the dialysis every 30 minutes until the patient's body temperature reaches 36.6° to 37.7° C (98° to 100° F).

Management

The body temperature should rise slowly, ideally no more than 1° F per hour. Because the response of the body to drugs is unpredictable, avoid administering drugs whenever possible, until the body temperature returns to normal. Complications observed during rewarming include DIC, cardiac dysrhythmias including cardiac arrest, pneumonia, pulmonary edema, CNS edema, ARDS, and renal failure.

HYPERTHERMIA AND HEAT-INDUCED ILLNESS (HEAT STROKE)

Heat stroke and heat-induced illness in dogs can be associated with excessive exertion, exposure to high environmental temperatures, stress, and other factors that cause an inability to dissipate heat. Brachycephalic breeds, obesity, laryngeal paralysis, and older animals with cardiovascular disease can be particularly affected. Hyperthermia is defined as a rectal temperature of 41° to 43° C (105° to 110° F). Clinical signs of hyperthermia include congested hyperemic mucous membranes, tachycardia, and panting. More severe clinical signs include collapse (heat prostration), ataxia, vomiting, diarrhea, hypersalivation, muscle tremors, loss of consciousness, and seizures. Heat-induced illness can affect all major organ systems in the body because of denaturation of cellular proteins and enzyme activities, inappropriate shunting of blood, hypotension, decreased oxygen delivery, and lactic acidosis. Cardiac dysrhythmias, interstitial and intracellular dehydration, intravascular hypovolemia, central nervous dysfunction, slough of gastrointestinal mucosa, oliguria, and coagulopathies can be seen as organ function declines. Excessive panting can result in respiratory alkalosis. Poor tissue perfusion results in a metabolic acidosis. Loss of water in excess of solutes such as sodium and chloride can lead to a free water deficit and severe hyponatremia. A marked increase in PCV occurs because of the free water loss. Severe abnormalities in electrolytes and pH can lead to cerebral edema and death.

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Immediate action

Treatment goals for the patient with heat-induced illness are to lower the core body temperature and support cardiovascular, respiratory, renal, gastrointestinal, neurologic, and hepatic functions. At the scene the veterinarian or caretaker can spray the animal with tepid (NOT COLD) water. Immersion in cold water or ice baths is absolutely contraindicated. Cold water and ice will cause extreme peripheral vasoconstriction, inhibiting the patient's ability to dissipate heat through conductive and convective cooling mechanisms. As a result, core body temperature will continue to rise despite the good intentions of well-doers at the scene. Animals that present to the veterinarian that have been cooled to the point of hypothermia have a worse prognosis. Once the animal has presented to the veterinarian, the goal is to cool the animal's body temperature with towels soaked in tepid water, cool intravenous fluids, and fans until the temperature has decreased to 103° F. Organ system monitoring and support is based on the severity and duration of the heat stroke and the ability of the body to compensate and respond to treatment.

Management

Management of the patient with heat-induced illness involves prompt aggressive cooling without being overzealous and creating iatrogenic hypothermia. Administer cool intravenous crystalloid fluids to replenish volume and interstitial hydration and correct the patient's acid-base and electrolyte abnormalities. Management consists of rule of twenty monitoring (See Rule of 20), taking care to evaluate, restore, and maintain a normal cardiac rhythm, blood pressure, urine output, and mentation. Administer antibiotics if there are any signs of gastrointestinal bleeding that will predispose the patient to bacterial translocation. Monitor baseline chemistry tests including a complete blood count, biochemical panel, platelet count, coagulation tests, and urinalysis. Treat coagulopathies including DIC aggressively and promptly (see also Disseminated Intravascular Coagulation). Severe changes in mentation including stupor or coma worsen a patient's prognosis. Following initial therapy, monitor the patient for a minimum of 24 to 48 hours for secondary organ damage, including renal failure, myoglobinuria, cerebral edema, and DIC. Dogs that are going to die of heat-induced illness usually die within the first 24 hours. Animals that survive longer than 24 hours have a more favorable prognosis.

Additional Reading

- Ahn A: Approach to the hypothermic patient. In Bonagura JD, editor: *Current veterinary therapy XII. Small animal practice*, Philadelphia, 1995, WB Saunders.
- Dhupa N: Hypothermia in dogs and cats, *Compend Contin Educ Pract Vet* 17:61, 1995.
- Drobatz KJ, Macintire DK: Heat-induced illness in dogs: 42 cases (1976-1993), *J Am Vet Med Assoc* 209:1894, 1996.
- Garcia-Lacaze M, Kirby R, Rudloff E: Peritoneal dialysis: not just for renal failure, *Compend Contin Educ Pract Vet* 24(10):758-771, 2002.
- Hackett TB: Heat stroke. In Wingfield WE, editor: *Veterinary emergency medicine secrets*, ed 2, Philadelphia, 2001, Hanley & Belfus.
- Oncken AK, Kirby R, Rudloff E: Hypothermia in critically ill dogs and cats, *Compend Contin Educ Pract Vet* 23(6):506-520, 2001.
- Walton RS: Hypothermia. In Wingfield WE, editor: *Veterinary emergency medicine secrets*, ed 2, Philadelphia, 2001, Hanley & Belfus.

MALIGNANT HYPERTHERMIA

Malignant hyperthermia is a syndrome that involves impaired muscular calcium metabolism. Malignant hyperthermia has been recognized as a consequence of exertion in Labrador Retrievers and in sensitized animals placed under anesthesia. Clinical signs of malignant hyperthermia are severe muscle spasm or fasciculation, unstable blood pressure, metabolic or respiratory acidosis, and a rapidly increased end-tidal carbon dioxide under anesthesia. The patient's temperature often rises above 42° C. Cellular death can result if the malignant hyperthermia is not recognized and treated rapidly.

Immediate action/treatment

Immediate treatment consists of cooling the patient with cooling measures as for hyperthermia and heat-induced illness (see the previous discussion), and eliminating the cause (i.e., exertion, anesthesia, or neuromuscular blockers such as succinylcholine). If the patient is under general anesthesia, hyperventilate the patient to help eliminate carbon dioxide and respiratory acidosis. Administer dantrolene sodium (1 to 2 mg/kg IV) to stabilize the sarcoplasmic reticulum and decrease its permeability to calcium.

Management

Animals with malignant hyperthermia should avoid any predisposing factors, including exertion, hyperthermia, and anesthesia. After an episode of malignant hyperthermia, administer crystalloid fluids intravenously to aid in the elimination of myoglobin. Monitor renal function closely for myoglobinuria and pigment damage to the renal tubular epithelium. Monitor and correct acid-base and electrolyte changes.

Additional Reading

Walters JM: Hyperthermia. In Wingfield WE, editor: *The veterinary ICU book*, Jackson, Wyo, 2001, Teton Newmedia.

SNAKEBITE: NONPOISONOUS

Sometimes it is difficult to assess whether an animal has been bitten by a poisonous or nonpoisonous snake. In Colorado, the bull snake closely resembles the prairie rattlesnake. Both snakes make similar noise and can be alarming if noticed on a hike or in the backyard. Whenever possible, identify the offending reptile but NEVER RISK BEING BITTEN. Know what types of venomous creatures are in the geographic area of the practice.

If an animal has been bitten by a nonpoisonous snake, usually the bite marks are small with multiple small tooth punctures, and the bite is relatively nonpainful. Usually local reaction is negligible. However, large boas or pythons also can inflict large crushing injuries that can cause severe trauma, including bony fractures.

Treatment for a nonpoisonous snakebite involves clipping the bite wound and carefully cleaning the area with antimicrobial scrub solution. Broad-spectrum antibiotics (e.g., amoxicillin-clavulanate, 16.25 mg/kg PO q12h) are indicated because of the extensive bacterial flora in the mouths of snakes. Monitor all snakebite victims for a minimum of 8 hours after the incident, particularly when the species of the offending reptile is in question. If clinical signs of envenomation occur, modify the patient's treatment appropriately and aggressively.

SNAKEBITE: POISONOUS

The two major groups of venomous snakes in North America are the pit viper and the coral snake. All venomous snakes are dangerous. The severity of any given bite depends on the toxicity of the venom, the amount of venom injected, the site of envenomation, the size of the animal bitten, and the time from bite/envenomation to seeking appropriate medical intervention.

PIT VIPER ENVENOMATION

The majority of reptile envenomations in the United States are inflicted by pit vipers, including the water moccasin (cottonmouth), copperhead, and numerous species of rattlesnakes. Pit vipers are characterized by a deep pit located between the eye and nostril, elliptic pupils, and retractable front fangs (Figure 1-45).

Localized clinical signs of pit viper envenomation may include the presence of bleeding puncture wounds, local edema close to puncture wounds, immediate severe pain or collapse, edema, petechiae, and ecchymosis with subsequent tissue necrosis. Systemic signs of pit viper envenomation may include hypotension, shock, coagulopathies, lethargy, weakness, muscle fasciculations, lymphangitis, rhabdomyolysis, and neurologic signs including respiratory depression and seizures. Neurologic signs largely are associated with envenomation

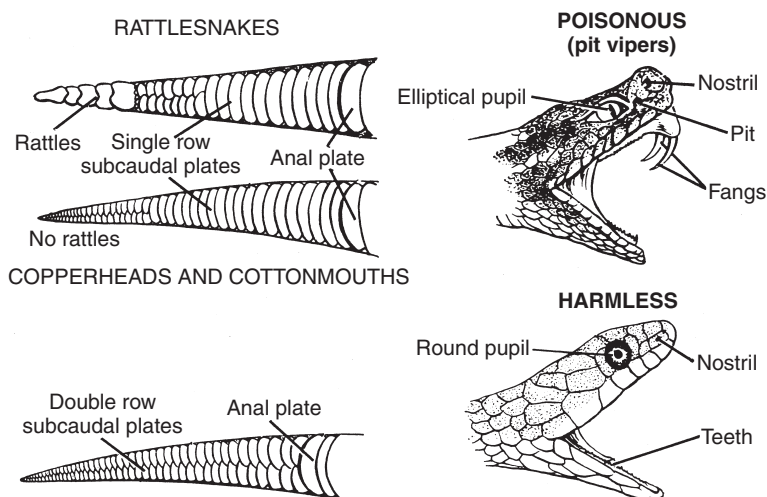


Figure 1-45: Characteristics of poisonous snakes.

(From Parrish HM, Carr CA: Bites by copperheads (*Ancistrodon contortrix*) in the United States. JAMA 201:927, 1967.)

by the Mojave and canebrake rattlesnakes, although a potent neurotoxin, Mojave toxin A, also has been identified in other subspecies of rattlesnake.

Clinical signs of envenomation may take several hours to appear. Hospitalize all suspected victims and monitor them for a minimum of 24 hours. The severity of envenomation cannot be judged solely on the basis of local tissue reaction. First aid measures by animal caretakers do little to prevent further envenomation. The most important aspect of initiating therapy is to transport the animal to the nearest veterinary emergency facility.

Immediate action

To determine whether an animal has been envenomated by a pit viper, examine a peripheral blood smear for the presence of echinocytes. Echinocytes will appear within 15 minutes of envenomation and may disappear within 48 hours. Other treatment should be initiated as rapidly and aggressively as possible, although controversy exists whether some therapies are warranted. The mainstay of therapy is to improve tissue perfusion with intravenous crystalloid fluids, prevent pain with judicious use of analgesic drugs, and when necessary, reverse or negate the effects of the venom with antivenin. Because pit viper venom consists of multiple fractions, treat each envenomation as a complex poisoning.

Obtain vascular access and administer intravenous crystalloid fluids (one fourth of a calculated shock dose) according to the patient's perfusion parameters of heart rate, blood pressure, and capillary refill time (see also Shock and Fluid Therapy). Opioid analgesics are potent and should be administered at the time of presentation. (See also Pharmacologic Means to Analgesia: Major Analgesics).

Diphenhydramine (0.5 to 1 mg/kg IM or IV) also can be administered to decrease the effects of histamine. Famotidine, a histamine₂ receptor antagonist, also can be administered (0.5 to 1 mg/kg IV) to work synergistically with diphenhydramine. Although antihistamines have no effect on the venom per se, they may have an effect on the tissue reaction to the venom and may prevent an adverse reaction to antivenin. The use of glucocorticosteroids is controversial. Glucocorticosteroids (dexamethasone sodium phosphate [Dex-SP], 0.25 to 0.5 mg/kg IV) may stabilize cellular membranes and inhibit phospholipase, an active component of some pit viper toxins.

Polyvalent antivenin is necessary in many cases of pit viper envenomation, except in most cases of prairie rattlesnake (*Crotalus viridis viridis*) envenomation in Colorado.

A recent study demonstrated no difference in outcome with or without the use of antivenin in cases of prairie rattlesnake envenomation. Clinically, however, patients that receive antivenin are more comfortable and leave the hospital sooner than those that do not receive antivenin. The exact dose of antivenin is unknown in small animal patients. Administer a dose of at least 1 vial of antivenin to neutralize circulating venom. Mix antivenin with a swirling, rather than a shaking motion, to prevent foaming. Mix the antivenin with a 250-mL bag of 0.9% saline, and then administer it slowly over a period of 4 hours. Pretreat animals with diphenhydramine (0.5 to 1 mg/kg IM) before the administration of antivenin, and then monitor the animal closely for clinical signs of angioneurotic edema, urticaria, tachyarrhythmias, vomiting, diarrhea, and weakness during the infusion. Administration of antivenin into the bite site is relatively contraindicated and ineffective because uptake is delayed, and systemic effects are the more life-threatening.

Management

Management of pit viper envenomation largely involves maintenance of normal tissue perfusion with intravenous fluids, decreasing patient discomfort with analgesia, and negating circulating venom with antivenin. Hydrotherapy to the affected bite site with tepid water is often soothing to the patient. The empiric use of antibiotics is controversial but is recommended because of the favorable environment created by a snakebite (i.e., impregnation of superficial gram-positive bacteria and gram-negative bacteria from the mouth of the snake into a site of edematous necrotic tissue). Administer amoxicillin-clavulanate (16.25 mg/kg PO q12h, or cephalexin, 22 mg/kg PO q8h). Also consider administration of NSAIDs (carprofen, 2.2 mg/kg PO q12h). Monitor the patient closely for signs of local tissue necrosis and the development of thrombocytopenia and coagulopathies including DIC (see Management of Disseminated Intravascular Coagulation). Treat coagulopathies aggressively to prevent end-organ damage.

CORAL SNAKE ENVENOMATION

Coral snakes are characterized by brightly colored bands encircling the body, with red and black separated by yellow. “Red on black, friend of Jack; red on yellow, kill a fellow.” Types of coral snakes include the Eastern coral, Texas coral, and Sonoran coral snakes. Clinical signs of coral snake envenomation may include small puncture wounds, transient initial pain, muscle fasciculations, weakness, difficulty swallowing/dysphagia, ascending lower motor neuron paralysis, miotic pinpoint pupils, bulbar paralysis, respiratory collapse, and severe hemolysis. Clinical signs may be delayed for as long as 18 hours after the initial bite.

Immediate therapy

Immediate treatment with antivenin is necessary in cases of coral snake envenomation before the clinical signs become apparent, whenever possible. Support respiration during paralysis with mechanical ventilation. Secure the patient’s airway with a cuffed endotracheal tube to prevent aspiration pneumonia.

Management

Clinical signs will progress rapidly once they develop. Rapid administration with antivenin is the mainstay of therapy in suspected coral snake envenomation. Respiratory and cardiovascular support should occur with mechanical ventilation and intravenous crystalloid fluids. Keep the patient warm and dry in a quiet place. Turn the patient every 4 to 6 hours to prevent atelectasis and decubitus ulcer formation. Maintain cleanliness using a urinary catheter and closed urinary collection system. Perform passive range of motion and deep muscle massage to prevent disuse atrophy of limb muscles and function. Treat aspiration pneumonia aggressively with broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6h, with enrofloxacin, 10 mg/kg IV q24h, and then change to oral once tolerated and the patient is able to swallow) for 2 weeks past the resolution of radiographic signs of pneumonia, intravenous fluids, and nebulization with sterile saline and coupage chest physiotherapy. Several weeks may elapse before a complete recovery.

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Additional Reading

- Brown DE, Meyer DJ, Wingfield WE, et al: Echinocytosis associated with rattlesnake envenomation in dogs, *Vet Pathol* 31:654-657, 1996.
- Fogel JE: Pit viper envenomation in dogs, *Stand Care Emerg Crit Care Med* 6(8):1-5, 2004.
- Hackett TB, Wingfield WE, Mazzaferro EM, et al: Clinical findings associated with prairie rattlesnake bites in dogs: 100 cases (1989-1998), *J Am Vet Med Assoc* 220(11):1675-1680, 2002.
- Kremer KA, Schaer M: Coral snake (*Micrurus fulvius fulvius*) envenomation in five dogs: present and earlier findings, *J Vet Emerg Crit Care* 5(1):9-15, 1995.
- Peterson P: Treating pit viper bites, *Vet Med* 93(10):885-890, 1998.

BLACK WIDOW SPIDER BITE

The adult black widow spider (*Latrodectus* spp.) can be recognized by a red to orange hour-glass-shaped marking on the underside of a globous, shiny, black abdomen. The immature female can be recognized by a colorful pattern of red, brown, and beige on the dorsal surface of the abdomen. Adult and immature females are equally capable of envenomation. The male is unable to penetrate the skin because of its small size. Black widow spiders are found throughout the United States and Canada. Black widow spider venom is neurotoxic and acts presynaptically, releasing large amounts of acetylcholine and norepinephrine. There appears to be a seasonal variation in the potency of the venom, lowest in the spring and highest in the fall. In dogs, envenomation results in hyperesthesia, muscle fasciculations, and hypertension. Muscle rigidity without tenderness is characteristic. Affected animals may demonstrate clinical signs of acute abdominal pain. Tonic-clonic convulsions may occur but are rare. In cats, paralytic signs predominate and appear early as a ascending lower motor neuron paralysis. Increased salivation, vomiting, and diarrhea may occur. Serum biochemistry profiles often reveal significant elevations in creatine kinase and hypocalcemia. Myoglobinemia and myoglobinuria can occur because of extreme muscle damage.

Management

Management of black widow spider envenomation should be aggressive in the cat and dog, particularly when the exposure is known. In many cases, however, the diagnosis is made based on clinical signs, biochemical abnormalities, and lack of other apparent cause. Antivenin (one vial) is available and should be administered after pretreatment with diphenhydramine. If antivenin is unavailable, administer a slow infusion of calcium-containing fluid such as lactated Ringer's solution with calcium gluconate while carefully monitoring the patient's ECG.

BROWN SPIDER BITE

Fiddleback, brown recluse, Arizona brown, *Loxosceles* spp.

The small brown nonaggressive spider is characterized by a violin-shaped marking on the cephalothorax. The neck of the violin points toward the abdomen. Brown spiders are found primarily in the southern half of the United States but have been documented as far north as Michigan. The venom of the brown spider has a potent dermatonecrotic effect and starts with a classic bull's-eye lesion. The lesion then develops into an indolent ulcer into dependent tissues promoted by complement fixation and influx of neutrophils into the affected area. The ulcer can take months to heal and often leaves a disfiguring scar. Systemic reactions are rare but can include hemolysis, fever, thrombocytopenia, weakness, and joint pain. Fatalities are possible.

Management

Immediate management of an animal with brown spider envenomation is difficult because there is no specific antidote and because clinical signs may be delayed until necrosis of the skin and underlying tissues becomes apparent through the patient's fur 7 to 14 days after the initial bite. Dapsone has been recommended at a dose of 1 mg/kg for 14 days. Surgical excision of the ulcer may be helpful if performed in the early stages of wound appearance.

Glucocorticosteroids may be of some benefit if used within 48 hours of the bite. The ulcer should be left to heal by second intention. Deep ulcers should be treated with antibiotics.

Additional Reading

Forrester MB, Stanley SK: Black widow spider and brown recluse spider bites in Texas from 1998-2002, *Vet Hum Toxicol* 45(5):270-273, 2003.

Twedt DC, Cuddon PA, Horn TW: Black widow spider envenomation in a cat, *J Vet Intern Med* 13(6):63-616, 1999.

OTHER POISONOUS CREATURES

Bufo species toxicosis

Bufo toad species (*B. marinus*, aka cane toad, marine toad, giant toad; and the Colorado River toad or Sonoran desert toad *B. alvarius*) can be associated with severe cardiac and neurotoxicity if an animal licks its skin. The severity of toxicity depends largely on the size of the dog. Toxins in the cane toad, *B. marinus*, include catecholamines and vasoactive substances (epinephrine, norepinephrine, serotonin, dopamine) and *bufo* toxins (bufagins, bufotoxin, and bufotenine), the mechanism of which is similar to cardiac glycosides. Clinical signs can range from ptialism, weakness, ataxia, extensor rigidity, opisthotonus, and collapse to seizures. Clinical signs associated with *B. alvarius* toxicity are limited largely to cardiac dysrhythmias, ataxia, and salivation.

Immediate action

The animal should have its mouth rinsed out thoroughly with tap water even before presentation to the veterinarian. If the animal is unconscious or actively seizing and cannot protect its airway, flushing the mouth is contraindicated. Once an animal presents to the veterinarian, the veterinarian should place an intravenous catheter and monitor the patient's ECG and blood pressure. Attempt seizure control with diazepam (0.5 mg/kg IV) or pentobarbital (2 to 8 mg/kg IV to effect). Ventricular dysrhythmias can be controlled first with esmolol (0.1 mg/kg). If esmolol is ineffective, administer a longer-acting parenteral β -antagonist such as propranolol (0.05 mg/kg IV). Ventricular tachycardia also can be treated with lidocaine (1 to 2 mg/kg IV, followed by 50 to 100 μ g/kg/minute IV CRI).

Management

Case management largely depends on supportive care and treating clinical signs as they occur. Monitor baseline acid-base and electrolyte balance because severe metabolic acidosis may occur that should be treated with intravenous fluids and sodium bicarbonate (0.25 to 1 mEq/kg IV). Monitor ECG, blood pressure, and mentation changes closely. Control seizures and cardiac dysrhythmias.

Additional Reading

Eubig PA: *Bufo* species intoxication: big toad, big problem, *Vet Med* 96(8):594-599, 2001.

Roberts BE, Aronson MG, Moses BL, et al: *Bufo marinus* intoxication in dogs: 94 cases (1997-1998), *J Am Vet Med Assoc* 216(12):1941-1944, 2000.

Gila monster (*Heloderma suspectum*) and Mexican bearded lizard (*Heloderma horridum*) bites

Lizards of the family Hemodermatidae are the only two poisonous lizards in the world. They are found in the Southwestern United States and Mexico. The venom glands are located on either side of the lower jaw. Because these lizards are typically lethargic and nonaggressive, bite wounds are rare. The lizards have grooved teeth that introduce the venom with a chewing motion as the lizard holds tenaciously to the victim. The majority of affected dogs are bitten on the upper lip, which is very painful.

Management

There are no proven first aid measures for bites from Gila monsters or Mexican bearded lizards. The lizard can be disengaged by inserting a prying instrument in between the jaws

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and pushing at the back of the mouth. The teeth of the lizard are brittle and break off in the wound. Topical irrigation with lidocaine and probing with a needle will aid in finding and removing the teeth from the victim. Bite wounds will bleed excessively. Irrigate wounds with sterile saline or lactated Ringer's solution, and place compression on the affected area until bleeding ceases. Monitor the patient for hypotension. Establish intravenous access, and administer intravenous fluids according to the patient's perfusion parameters. Antibiotic therapy is indicated because of the bacteria in the lizard's mouth. Because no antidote is available, treatment is supportive according to patient signs.

FRACTURES AND MUSCULOSKELETAL TRAUMA

The majority of musculoskeletal emergencies are the result of external trauma, most commonly from motor vehicle accidents. Blunt trauma invokes injury to multiple organ systems as a rule, rather than an exception. Because of this, massive musculoskeletal injuries are assigned a relatively low priority during the initial triage and treatment of a traumatized animal. Perform a rapid primary survey and institute any lifesaving emergency therapies. Adhere to A CRASH PLAN or the ABCs of resuscitation (see Initial Emergency Examination, Management, and Triage).

Although musculoskeletal injuries are assigned a relatively lower priority, the degree of recovery from these injuries and financial obligation for fracture repair sometimes becomes a critical factor in a client's decision whether to pursue further therapy. One of the most important deciding factors is the long-term prognosis for the patient to have a good quality of life following fracture repair.

The initial management of musculoskeletal injuries is important in ensuring the best chance for maximal recovery with minimal complications after definitive surgical fracture repair. This is particularly important for open fractures, spinal cord compromise, multiple fractures, open joints, articular fractures, physeal fractures, and concomitant ligamentous or neurologic compromise (Box 1-41).

IMMEDIATE ACTION

Immediately after the initial primary survey of a patient, perform a more thorough examination, including an orthopedic examination. Multiple injuries often are observed in the patient that falls from height (e.g., "high-rise syndrome"), motor vehicle accidents, gunshot wounds, and encounters with other animals (e.g., "big-dog-little-dog"). Address the most life threatening injuries, and palliate musculoskeletal injuries until more definitive repair can be attempted when the patient is more stable.

In animals with the history of potential for multiple injuries, search thoroughly and meticulously for areas of injury to the spinal column, extremities, and for small puncture wounds. Helpful signs that can provide a clue as to an underlying injury include swelling,

BOX 1-41 CLASSIFICATION OF SKELETAL TRAUMA

GROUP I: CRITICAL

Immediate therapy needed within a few hours

Examples: Compressive skull fractures, spine fracture or luxation/subluxation, open fractures or luxations

GROUP II: SEMICRITICAL

Early treatment within 2 to 5 days

If definitive repair is not attempted within 2 to 5 days, complications including delayed healing and poor long-term results may occur.

Examples: Articular fractures, physeal fractures, joint luxation/subluxation, slipped capital epiphysis

GROUP III: NONCRITICAL

Delayed treatment (within several days)

Scapular and pelvic fractures, greenstick fractures, closed long bone fractures

bruising, abnormal motion, and crepitus (caused by subcutaneous emphysema or bony fracture). If the patient is alert, look for areas of tenderness or pain. In unconscious or depressed patients, reexamine the patient after the patient becomes more mentally alert. Injuries often are missed during the initial examination in obtunded patients because of the early response and attenuation of pain. Unconscious or immobile patients must have radiographic examination of the spinal column following stabilization and support. Palpate the skull carefully for obvious depressions or crepitus that may be associated with a skull fracture. Localization of the injury can be determined by motion in abnormal locations, swelling caused by hemorrhage or edema, pain during gentle movement or palpation, deformity, angular change, or a significant increase or decrease in normal range of motion of bones and joints. Perform a rectal examination in all cases to palpate for pelvic fractures and displacement.

Once the diagnosis of a fracture or luxation has been confirmed, look for any evidence of skin lacerations or punctures near the fracture site. In long-haired breeds, clipping the fur near the fracture site often is necessary to perform a thorough examination of the area. If any wounds are found, the fracture is classified as an open fracture until proven otherwise. In some cases, the open fracture is obvious, with a large section of bone fragment protruding through the skin. In other cases, the puncture wound may be subtle, with only a small amount of blood or pinpoint hole in the skin surface. Characteristics observed with open fractures include bone penetration, fat droplets or marrow elements in blood coming from the wound, subcutaneous emphysema on radiographs, and lacerations in the area of a fracture. Protect the patient from further injury or contamination of wounds. Excessive palpation to intentionally produce crepitus is inappropriate because it causes severe patient discomfort and has the potential to cause severe soft tissue and neurologic injury at the fracture site. Sedation and analgesia aids in making the examination more comfortable for the patient and allows localization of the injury and comparison with the opposite extremity. Higher-quality radiographs can be performed to determine the extent of the injury when the animal is sedated adequately and pain is controlled.

INITIAL FRACTURE MANAGEMENT

Sedate the patient judiciously with analgesic drugs. Opioid drugs work well for orthopedic pain, produce minimal cardiorespiratory depression, and can be reversed with naloxone if necessary. Handle the fracture site gently to avoid causing further pain and soft tissue injury at the fracture site. Rough or careless handling of a fracture site can cause a closed fracture to penetrate through the skin and become an open fracture. Cover open fractures immediately to prevent contamination of the fracture with nosocomial infection from the hospital. Administer a first-generation cephalosporin (cephalexin, 22 mg/kg PO q8h, or cefazolin, 22 mg/kg IV q8h). The bandage also serves to control hemorrhage and prevent desiccation of the bones and surrounding soft tissue structures. Leave the initial bandages in place until the patient's cardiorespiratory status has been determined to be stable and more definitive wound management can occur in a clean, preferably sterile location.

Examine the neurologic status and cardiovascular status of the limb before and after treatment. Determine the vascular status of the limb by checking the color and temperature of the limb, the state of distal pulses, and the degree of bleeding from a cut nail bed. In patients with severe cardiovascular compromise and hypotension caused by hemorrhagic shock, the viability of the limb may be in question until the cardiovascular status and blood pressure are normalized. Reduction of the fracture or straightening of gross deformities may return normal vascularity to the limb. When checking neurologic status, examine for motor and sensory function to the limb. Swelling may increase pressure on the nerves as they run through osteofascial compartments, resulting in decreased sensory or motor function, or neurapraxia. Diminished function often returns to normal once the swelling subsides. Serial physical examinations in the patient and response to initial stabilization therapy can lead to a higher index of suspicion that more occult injuries are present, such as a diaphragmatic hernia, perforated bowel, lacerated liver or spleen, or uroabdomen.

To prevent ongoing trauma, reduce any fracture and then stabilize the site above and below the fracture. A modified Robert Jones splint or bandage often works well for fractures

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involving the distal extremities. Fractures of the humerus or femur are difficult to immobilize without the use of spica or over-the-hip coaptation splints to prevent mobility. Inappropriate bandaging of humerus or femur fractures can result in a fulcrum effect and worsen the soft tissue and neurologic injuries.

Further displacement of vertebral bodies or luxations can cause cord compression or laceration such that return to function becomes impossible. Immediately place any patient with a suspected spinal injury on a flat surface, and tape down the animal to prevent further movement until the spine has been cleared by a minimum of two orthogonal radiographic views (lateral and ventrodorsal views performed as a cross-table x-ray technique).

OPEN MUSCULOSKELETAL INJURY

Wounds associated with musculoskeletal trauma are common and include injury to the bones, joints, tendons, and surrounding musculature (Box 1-42). Major problems associated with these cases are the presence of soft tissue trauma that makes wound closure hazardous or impossible, because of the risk of infection. Chronic deep infection of traumatized wounds can cause delayed healing and sequestrum to develop, particularly if there is avascular bone or cartilage within the wound.

In the early management of an open fracture, the areas should be splinted without pulling any exposed bone back into the soft tissue. The wound should not be probed or soaked, as nosocomial bacteria and other external contaminants can be introduced into the wound, leading to severe infection. Because of the risk of actually causing infection, probing, flushing, or replacing tissues back into the wound should be performed at the time of formal debridement when the patient is physiologically stable. Immediate bactericidal antibiotic therapy with a first-generation cephalosporin should be started immediately to obtain adequate concentrations of antibiotics at the fracture site. The duration of antibiotic therapy should ideally be limited to 2–3 days to prevent the risk of superinfection.

Treatment

Treatment of open musculoskeletal injury involves three considerations: initial inspection and wound debridement, stabilization and repair, and wound bandaging.

BOX 1-42 CLASSIFICATION OF OPEN WOUNDS BY DEGREE OF SOFT TISSUE INJURY

TYPE I WOUND

Minimal soft tissue trauma and devitalization

When associated with a fracture, wound is created from the inside out by penetration of bone fragments through the skin or from a low-energy gunshot.

Simple or comminuted fracture pattern

Good stability of the two main bone segments

Treatment and prognosis are good and similar to those of a closed injury if wound is debrided and stabilized within 6 to 8 hours.

TYPE II WOUND

Moderate soft tissue contusion and devitalization

When associated with a fracture, wound is created from the outside in.

Major deep injury with considerable soft tissue stripping from bone and muscle damage

Simple or comminuted fracture pattern

Prognosis is good if wound is debrided within 6 hours of injury and provided rigid stabilization with a bone plate or external fixator.

TYPE III WOUND

Results from major external force

Severe damage and necrosis of skin, subcutaneous tissue, muscle, nerve, bone, tendon, and arteries

Soft tissue damage may vary from crush injury to shearing injury associated with bite wounds or low-speed automobile accidents.

Requires immediate and delayed sequential debridement and rigid external fixation

Can require prolonged healing times

Guarded prognosis

Initial inspection and wound debridement include the following steps:

1. After the patient's cardiovascular status has been stabilized and it has been determined that it can withstand anesthesia, place the animal under general anesthesia and remove the temporary splint.
2. Keeping the wound covered, shave the surrounding fur.
3. Remove the covering and then place sterile lubricant jelly over the wound. Shave the fur to the edges of the wound margin.
4. Wash away any entrapped fur and the lubricant jelly.
5. Complete an antiseptic scrub of the surrounding skin.
6. If the wound is a small puncture (e.g., gunshot pellets or bites), probe the wound with a sterile hemostat. Do a thorough debridement if tissues deep to the hole are cavitated. If not deep, create a hole for drainage.
7. Flush the wound with a physiologic solution (lactated Ringer's solution is preferred).
8. Debride the wound from outward to inward. Cut away damaged areas of skin and deeper tissues to open up underlying cavitations and tissue injury.
9. Continuously irrigate with warm physiologic solution (lactated Ringer's solution is preferred). The stream must be strong enough to flush debris out of the bottom of the wound. To accomplish this, attach a 20-gauge needle to a 35-mL syringe (will deliver 7 psi). Excise any obviously devitalized tissue.
10. Do not remove any bone fragments that are firmly attached to soft tissue. Do not cut into healthy soft tissue to find bullet or bone fragments, unless the bullet can cause injury to joints or nerve tissue.
11. Do a primary repair of tendons and nerves if the wound is type I and recent (within 8 hours of the initial injury). If the wound is too severe or if there is obvious infection, tag the ends of the tendons and nerves for later repair.

It is best to stabilize and repair open fractures as soon as the patient's cardiovascular and respiratory status can tolerate general anesthesia, provided that adequate stabilization is possible. If this is not possible because of the level of experience of the surgeon or the lack of necessary equipment, it is best to perform wound management and place a temporary splint until definitive repair can be performed.

Wound bandaging is discussed in the section on Bandaging Techniques.

ARTICULAR CARTILAGE INJURY

Structural injuries to the joints are common and can involve both ligaments and articular cartilage injuries. Cartilage does not heal well; therefore, injuries involving articular cartilage can lead to a significant loss of function and degenerative joint disease (osteoarthritis). Cartilage injuries that are superficial evoke a short-lived enzymatic and metabolic response that does not stimulate enough cellular growth to repair the defect. Superficial lesions remain as defects but do not progress to chondromalacia or osteoarthritis. Deep cartilage lacerations that extend to subchondral bone produce an exuberant healing response from the cells of the underlying cartilage. In many cases, this material undergoes degeneration and leads to osteoarthritis. Impact injuries to surface cartilage can cause chondrocyte and underlying bone injury. These lesions rapidly progress to osteoarthritis; however, they may be totally or partially reversible.

LIGAMENTOUS INJURIES

Treatment of grade I injuries requires short-term coaptation splints and has a good prognosis. Grade II injuries require surgical treatment with a suture stent and consistent post-operative coaptation splints to heal and maintain good function. Healing of grade III injuries often is a problem, and suture stents or surgical reapproximation may be indicated. Failure to immobilize joints that are frequently flexed (elbow and stifle) can result in late complications of ligament repair. Ligamentous injuries of joints, particularly the collateral ligaments of the stifle, elbow, and hock, and carpal hyperextension injuries are commonly missed and may require surgical fixation, including arthrodesis (Box 1-43).

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BOX 1-43 CLASSIFICATION OF LIGAMENTOUS INJURIES

Grade I sprain: Rupture of a portion of the ligament with minimal lengthening. Preservation of anatomical and mechanical integrity.

Grade II sprain: A portion of the ruptured ligament is stretched. Ligament is longer but still intact.

Grade III sprain: Complete ligament disruption

FRACTURES IN THE IMMATURE ANIMAL

Fractures in immature animals differ from those in adults in that young puppies and kittens have a great ability to remodel bone. Remodeling is dependent on the age of the patient and the location of the fracture. The younger the puppy or kitten and the closer the fracture to the epiphysis or growth plate, the greater the potential for remodeling and the development of angular limb deformities. Remodeling occurs more effectively in long-limbed breeds of dogs than in short-limbed breeds. Fractures through the growth plate of immature animals may potentially cause angular limb deformities, joint dislocations or incongruity, and osteoarthritis. This form of injury is commonly observed in the distal ulnar growth plate and the proximal and distal radial growth plates.

HIGH-RISE SYNDROME

High-rise syndrome in cats is seen in cats that fall from a height usually greater than 30 feet. It occurs most frequently in high-rise buildings in urban areas where cats lie on window ledges and suddenly fall out the window. The most common lesions observed in cats that fall from heights are thoracic injuries (rib and sternal fractures, pneumothorax, and pulmonary contusions) and facial and oral trauma (lip avulsions, mandibular symphyseal fractures, fractures of the hard palate, and maxillary fractures). Limb and spinal cord fractures and luxations, radius and ulna fractures, abdominal trauma, urinary tract trauma, and diaphragmatic hernias are also common. The injuries sustained are often found in combination, rather than as an isolated injury of one area of the body.

Follow the mnemonic A CRASH PLAN when managing a cat suffering from high-rise syndrome, treating the animal immediately for shock. Following cardiovascular and respiratory stabilization, evaluate thoracic and abdominal radiographs, including those of the spine. Evaluate the bladder closely, making sure that the cat is able to urinate effectively. Examine the hard palate, maxilla, and mandibular symphysis for fractures. Palpate the pelvis and carefully manipulate all limbs to examine for fractures or ligamentous injuries. Finally, perform a complete neurologic examination. Patients that fall less than five stories often have a more guarded prognosis than patients that fall from higher levels.

Additional Reading

- Aron DN: Emergency management of the musculoskeletal trauma patient. In: Emergency medicine and critical care in practice. Veterinary Learning Systems, Trenton, NJ, 1992,.
- Gordon LE, Thacher C, Kapatkin A: High-rise syndrome in dogs: 81 cases. JAVMA 202(1): 118-122, 1993.
- Papazoglou LG, Galatos AD, Patsikas MN, et al: High-rise syndrome in cats: 207 cases (1988-1998) Aust Vet Pract 31(3):98-102, 2001.
- Vnuk D, Pirkic B, Maticic D, et al: Feline high-rise syndrome: 119 cases (1998-2001), J Feline Med Surg 6(5):301-312, 2004.

GASTROINTESTINAL EMERGENCIES**ORAL CAVITY**

Sometimes the owner witnesses the ingestion of a foreign body during play, such as throwing a stick or fetching a ball. Cats tend to play with string or thread that becomes caught around the base of the tongue. In many cases, however, ingestion of the foreign object is not witnessed, and diagnosis is made based on clinical signs and physical examination.

Foreign bodies lodged in the oral cavity often cause irritation and discomfort, including difficulty breathing and difficulty swallowing. Often, an animal paws at its mouth in an

attempt to dislodge a stick or bones wedged across the roof of the mouth. Irritation, inability to close the mouth, and blockage of the oropharynx can result in excessive drooling. The saliva may appear blood-tinged due to concurrent soft tissue trauma (Figs 1-46 and 1-47).

Obstruction of the glottis by a foreign body (e.g., tennis ball or toy) can result in cyanosis secondary to an obstructed airway and hypoxemia. In many cases, the object is small enough to enter the larynx but too large to be expelled. If a foreign object is lodged in the mouth for more than several days, halitosis and purulent discharge may be present.

Many animals are anxious at the time of presentation and may require sedation or a light plane of anesthesia to remove the foreign object. The animal may bite personnel and may have bitten the owner during his or her attempt to remove the object from the mouth en route to the hospital. Propofol (4 mg/kg IV) or a combination of propofol with diazepam (0.5-1 mg/kg IV) is an excellent combination for a light plane of anesthesia. Exercise caution when anesthetizing a patient with a ball lodged in the airway, as further compromise of respiratory function may occur and cause worsening of the hypoxemia.

Before inducing anesthesia, assemble all supplies necessary to remove the object. Make sure that rigid towel clamps, sponge forceps, and bone forceps are on hand, because the foreign object is often very slippery with saliva. Hemostats and carmalts may slip and not be useful in the removal of the foreign object.

Place a peripheral intravenous catheter to secure vascular access prior to anesthetic induction. Have available the supplies necessary for an emergency tracheostomy, if the foreign object cannot be removed by usual methods. Induce a light plane of anesthesia and then grasp the object with the sponge forceps or towel clamps, and extract. Monitor the cardiorespiratory status of the animal at all times during the extraction process. If you are



Figure 1-46: Excessive ptyalism and gagging or excessive swallowing should increase suspicion of the presence of an esophageal or pharyngeal foreign body.

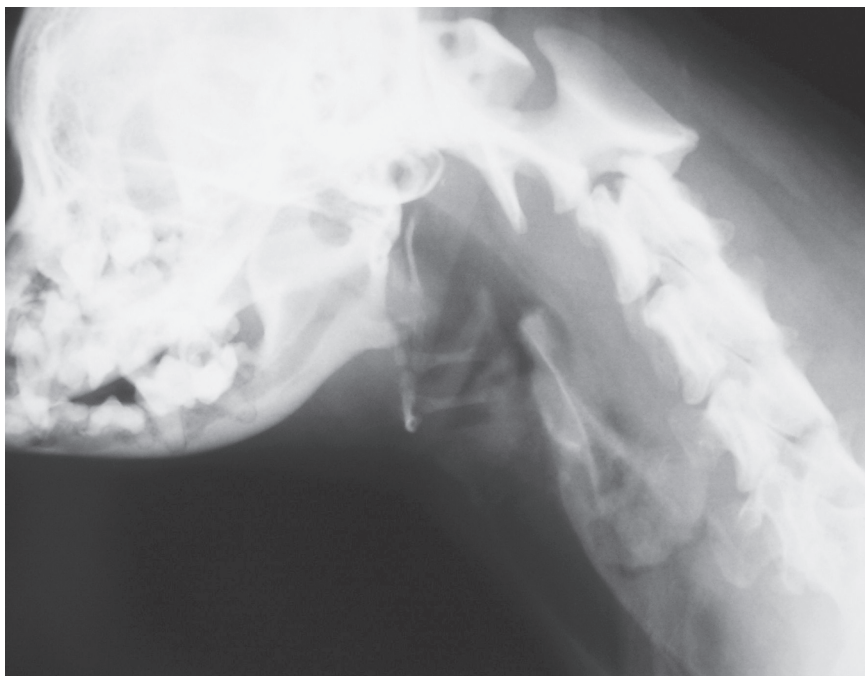


Figure 1-47: Radiograph of a chicken bone lodged in the patient's pharynx.

unable to remove the object, and if severe respiratory distress, including cyanosis, bradycardia, or ventricular dysrhythmias, develop, perform a tracheostomy distal to the site of obstruction.

Once the foreign body has been removed, administer supplemental flow-by oxygen until the animal awakens. If laryngeal edema or stridor on inspiration is present, administer a dose of dexamethasone sodium phosphate (0.25 mg/kg IV, IM, SQ) to decrease inflammation. The patient should be carefully monitored for 24 hours, because noncardiogenic pulmonary edema can develop secondary to airway obstruction.

ESOPHAGEAL FOREIGN BODIES

Esophageal foreign bodies pose a serious medical emergency. It is helpful if the owner witnessed ingestion of the object and noted rapid onset of clinical signs. In many cases, however, ingestion is not witnessed, and the diagnosis must be made based on clinical signs, thoracic radiographs, and results of a barium swallow. The most common clinical signs are excessive salivation with drooling, gulping, and regurgitation after eating. Many animals will make repeated swallowing motions. Some animals exhibit a rigid “sawhorse” stance, with reluctance to move immediately after foreign body ingestion and esophageal entrapment.

After completing a physical examination, evaluate cervical and thoracic radiographs to determine the location of the esophageal obstruction. Esophageal foreign objects are lodged most commonly at the base of the heart, the carina, or just orad to the lower esophageal sphincter. If the object has been lodged for several days, pleural effusion and pneumomediastinum may be present secondary to esophageal perforation. Endoscopy is useful for both diagnosis and removal of the foreign object; however, it is invasive and requires general anesthesia (Fig. 1-48).

Remove foreign objects lodged in the esophagus with a rigid or flexible endoscope after the patient has been placed under general anesthesia. Evaluate the integrity of the esophagus both before and after removal of the material because focal perforation or pressure

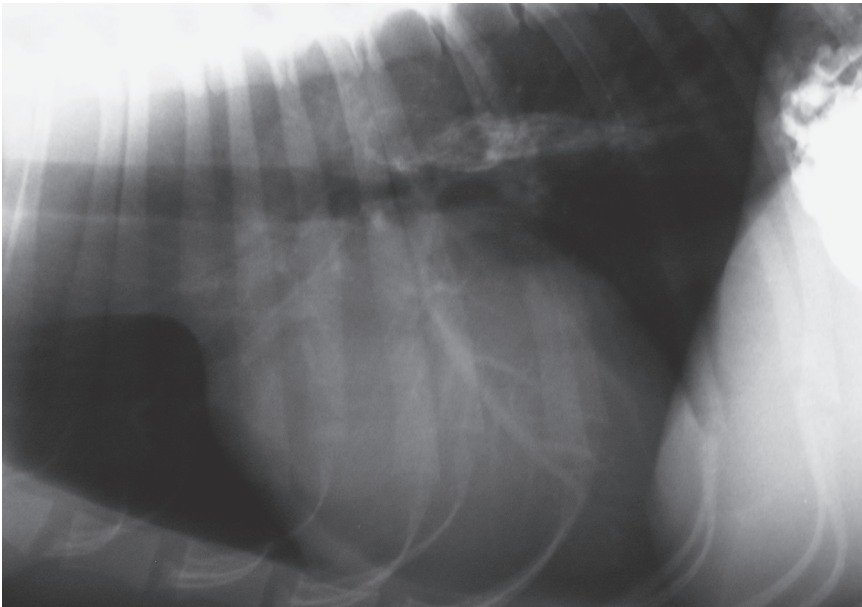


Figure 1-48: Example of an esophageal foreign body. Common locations are the carina and thoracic inlet.

necrosis can be present. Necrosis of the mucosa and submucosa of the esophagus often leads to stricture formation or perforation.

Attempt to retrieve the object with a flexible fiberoptic endoscope if available. Rigid tube endoscopy can also be performed. In many cases, smooth objects that cannot be easily grasped can be pushed into the stomach and allowed to dissolve or may be removed by gastrotomy. If the foreign body is firmly lodged in the esophagus and cannot be pulled or pushed into the stomach, or if perforation has already occurred, the prognosis for return to function without strictures is not favorable. In such cases, referral to a surgical specialist is recommended for esophagostomy or esophageal resection.

After removal of the object, carefully examine the esophagus and then administer gastroprotectant agents (famotidine, 0.5 mg/kg PO bid; sucralfate slurry, 0.5-1.0 g/dog) for a minimum of 5 to 7 days. To rest the esophagus, the patient should receive nothing per os (NPO) for 24 to 48 hours. If esophageal irritation or erosion is moderate to severe, a percutaneous gastrotomy tube should be placed for feeding until the esophagus heals. Perform repeat endoscopy every 7 days to evaluate the healing process and to determine whether stricture formation is occurring.

STOMACH

Persistent vomiting immediately or soon after eating is often associated with a gastric foreign body. In some cases, the owner knows that the patient has ingested a foreign body of some kind. In other cases, continued vomiting despite lack of response to conservative treatment (NPO, antiemetics, gastroprotectant drugs) prompts further diagnostic procedures, including abdominal radiographs and bloodwork. Obstruction to gastric outflow and vomiting of hydrochloric acid often cause a hypochloremic metabolic acidosis. Radiopaque gastric foreign bodies may be observed on plain films. Radiolucent cloth material may require a barium series to delineate the shape and location of the foreign body (Fig. 1-49).

Treatment consists of removal with flexible endoscopy or a simple gastrotomy. Most animals with uncomplicated gastric foreign bodies are relatively healthy, but any metabolic and electrolyte abnormalities should be corrected prior to anesthesia and surgery.

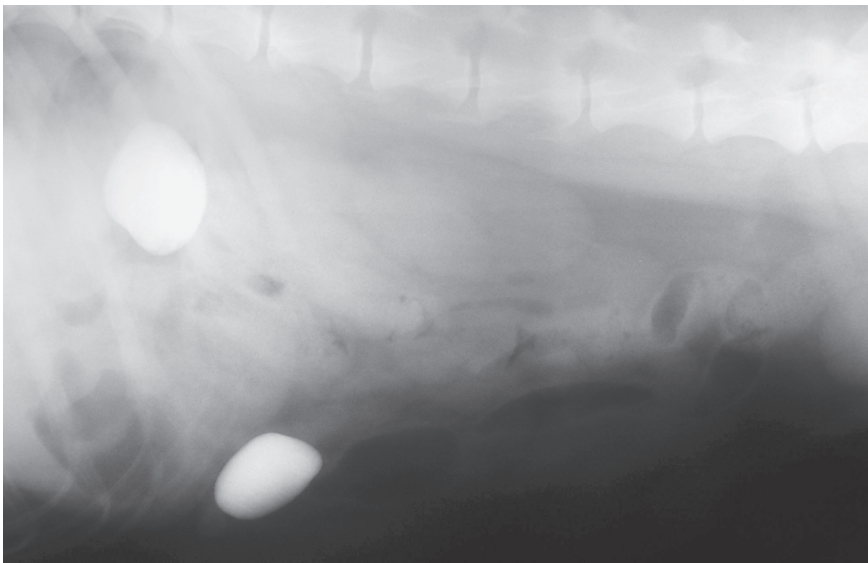


Figure 1-49: Lateral abdominal radiograph with two radiopaque densities within the lumen of the small intestine, consistent with rocks.

SMALL INTESTINAL OBSTRUCTION

Small intestinal obstruction can be caused by foreign bodies, tumors, intussusception, volvulus, or strangulation within hernias. Regardless of the cause, clinical signs of small intestinal obstruction depend on the location and degree of obstruction, and whether the bowel has perforated. Clinical signs associated with a high small intestinal obstruction are usually more severe and more rapid in onset compared with partial or complete obstruction of the jejunum or ileum. Complete obstructions that allow no fluid or chyme to pass are worse than partial obstructions, which can cause intermittent clinical signs interspersed with periods of normality (Table 1-36).

The most common clinical signs associated with a complete small intestinal obstruction are anorexia, vomiting, lethargy, depression, dehydration, and sometimes abdominal pain. Early clinical signs may be limited to anorexia and depression, making a diagnosis challenging unless the owner has a suspicion that the animal ingested some kind of foreign object. Obstructions cranial to the common bile duct and pancreatic papillae lead to vomiting of gastric contents, namely hydrochloric acid, and a hypochloremic metabolic alkalosis. Obstructions caudal to the common bile duct and pancreatic papillae result in loss of other electrolytes and sometimes mixed acid-base disorders.

TABLE 1 - 36 Localizing Signs for Patients with Bowel Obstruction						
Condition	Onset	Progression of vomiting	Frequency	Volume of vomit	Tenesmus	Abdominal distention
High small bowel	Rapid	Rapid	Frequent	Large volume	Absent	Absent
Low small bowel	Slower	Slower	Less frequent	Small volume	Diarrhea	Present
Large bowel	Subacute to chronic	Slow	Occasional	Scant	Often with diarrhea	Present



Figure 1-50: Intraoperative photograph depicting plication of the jejunum by a linear foreign body.

Eventually, all animals with small intestinal obstruction vomit and have fluid loss into dilated segments of bowel, leading to dehydration and electrolyte abnormalities. Increased luminal pressure causes decreased lymphatic drainage and bowel edema. The bowel wall eventually becomes ischemic and may rupture.

Linear foreign bodies should be suspected in any vomiting patient, particularly cats. String or thread often is looped around the base of the tongue and can be visualized in many cases by a thorough oral examination. To look properly under the tongue, grasp the top of the animal's head with one hand, and pull the lower jaw open with the index finger of the opposite hand while pushing up the thumb simultaneously on the tongue in between the intermandibular space. Thread and string can be observed lying along the ventral aspect of the tongue. In some cases, if a linear foreign body is lodged very caudally, it cannot be visualized without heavy sedation or anesthesia.

Linear foreign bodies eventually cause bowel obstruction and perforation of the intestines along the mesenteric border. The foreign material (e.g., string, thread, cloth, pantyhose) becomes lodged proximally, and the intestines become plicated as the body attempts to push the material caudally through the intestines (Fig. 1-50). Continued peristalsis eventually causes a sawing motion of the material and perforation of the mesenteric border of the intestines. Once peritonitis occurs, the prognosis is less favorable unless prompt and aggressive treatment is initiated.

Reevaluate any patient that does not respond to conservative symptomatic therapy, performing a complete blood count, serum biochemical panel (including electrolytes), and abdominal radiographs. Intestinal masses may be palpable on physical examination and are often associated with signs of discomfort or pain when palpating over the mass. Radiography and abdominal ultrasound are the most useful diagnostic aids. Plain radiographs may be diagnostic when the foreign object is radiodense or there is characteristic dilation or plication of bowel loops. As a rule of thumb, the width of a loop of small bowel should be no larger than twice the width of a rib. Diagnosis of small intestinal obstruction or ileus can be based on the appearance of stacking loops of dilated bowel. Comparison of the width of the bowel with the width of a rib is often performed. With mild dilation, the bowel width is three to four times the rib width; with extensive dilation, five to six times the rib width (Fig. 1-51). In cases of linear foreign bodies, C-areas

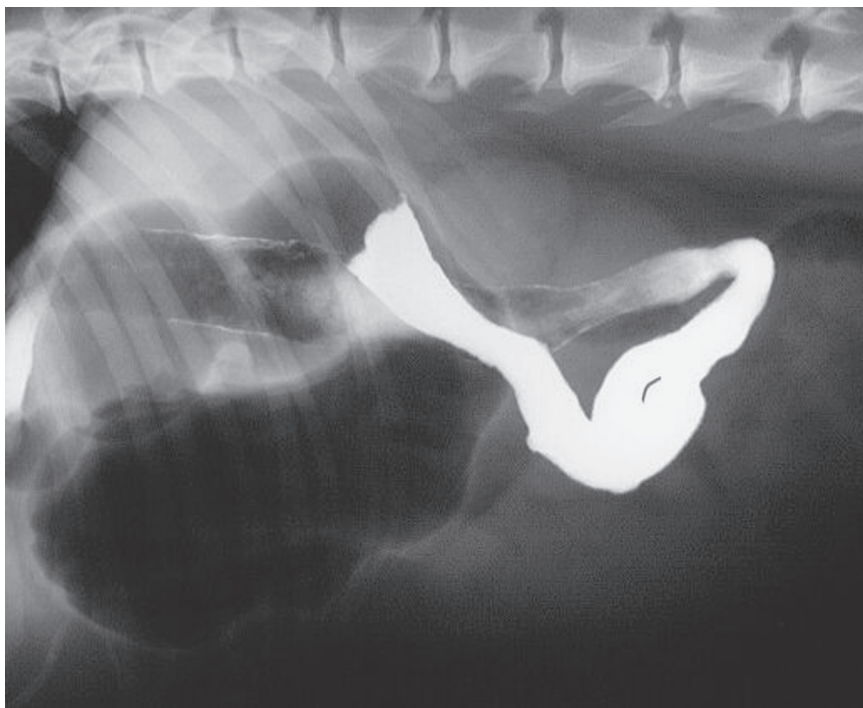


Figure 1-51: After 60 minutes, the barium has stopped moving and has reached a blunt, intraluminal intestinal foreign body. Note that barium appears wedge-shaped or square at the site of the foreign body.

(comma-shaped areas) of gas trapped in the plicated bowel will appear stacked on one another. Blunt, wedge-shaped areas of gas or square linear areas of gas adjacent to a distended bowel loop are characteristic of a foreign body lodged in the intestine. Contrast radiography is indicated when confirmation of the suspected diagnosis is necessary and ultrasonography is not available. Contrast material may outline the object or abruptly stop orad to the obstruction.

The definitive treatment of any type of small intestinal foreign body is surgical removal. Linear foreign bodies sometimes pass, but they should never be left untreated in a patient that is demonstrating clinical signs of inappetence, vomiting, lethargy, and dehydration. The timing of surgery is critical because the risk of intestinal perforation increases with time. Prior to surgery, correct any acid-base and electrolyte abnormalities with intravenous fluid therapy. Administer broad-spectrum antibiotics. Perform an enterotomy or intestinal resection and anastomosis as soon as possible once the patient's acid-base and electrolyte status have been corrected.

LARGE INTESTINAL FOREIGN BODIES

Clinical signs of a foreign body in the large bowel are usually nonexistent. In most cases, if a foreign object has passed successfully through the small bowel, it will pass through the large bowel without incident unless bowel perforation and peritonitis occur. Penetrating foreign bodies such as needles often cause localized or generalized peritonitis, abdominal pain, and fever. Hematochezia may be present if the foreign object causes abrasion of the rectal mucosa.

Symptomatic patients should have abdominal radiographs performed. Colonoscopy or exploratory laparotomy should be performed if survey radiographs are suggestive of a large intestinal obstruction or perforation. In most cases, large intestinal foreign bodies will pass without incident. Surgery is required to treat perforations, peritonitis, or abscesses.

RECTUM AND ANUS

Foreign bodies in the rectum and anus often are the result of ingestion of bones, wood material, needles, and thread, or malicious external insertion. Often the material can pass through the entire gastrointestinal tract and then get stuck in the anal ring. Clinical signs include hematochezia and dyschezia with straining to defecate. Diagnosis is made by visual examination of the item in the anus, or by careful digital palpation after heavy sedation or short-acting general anesthesia. Radiography is helpful in locating needles that have penetrated the rectum and lodged in the perirectal or perinatal tissues. Treatment consists of careful removal of the needle digitally or surgically.

ACUTE INTUSSUSCEPTION

Intussusception is the acute invagination of one segment of bowel (the *intussusceptum*) into another (the *intussusciens*). The proximal segment always invaginates into the distal segment of bowel. Intussusception most commonly occurs in puppies and kittens less than 1 year of age but can occur in an animal of any age with hypermotility of the small bowel, gastrointestinal parasites, and severe viral or bacterial enteritis. Intussusception occurs primarily in the small bowel in the jejunum, ileum, and ileocolic junction.

Clinical signs include vomiting, abdominal discomfort, and hemorrhagic diarrhea. Usually, hemorrhagic diarrhea is the first noticeable sign, and in puppies, may be due to parvoviral enteritis, with secondary intussusception. Usually, the obstruction is partial with mild clinical signs. More serious clinical signs develop as the obstruction becomes more complete. Differential diagnoses include hemorrhagic gastroenteritis, parvoviral enteritis, gastrointestinal parasites, intestinal foreign body, bacterial enteritis, and other causes of vomiting and diarrhea.

The diagnosis of intussusception is often made based on palpation of a sausage-shaped firm, tubular structure in the abdomen accompanied by clinical signs and abdominal pain. Plain radiographs may demonstrate segmental or generalized dilated segments of bowel, depending on the duration of the problem. Ultrasonographs of the palpable mass resemble the layers of an onion, with hyperechoic intestinal walls separated by less echogenic edema.

Treatment consists of correction of the patient's acid-base and electrolyte abnormalities with intravenous fluids and surgical reduction or removal of the intussusception with resection and anastomosis. Although enteroplication has been suggested, the technique has fallen out of favor because of the increased risk of later obstruction. The primary cause of intestinal inflammation and hypermotility must be identified and corrected.

GASTRIC DILATATION-VOLVULUS

Gastric dilatation can occur with or without volvulus in the dog. Gastric dilatation-volvulus (GDV) occurs primarily in large- and giant-breed dogs with deep chests, such as the Great Dane, Labrador Retriever, Saint Bernard, German Shepherd Dog, Gordon and Irish Setters, Standard Poodle, Bernese Mountain Dog, and Bassett Hound. The risk of GDV increases with age; however, it can be seen in dogs as young as 4 months. Deep, narrow-chested breeds are more likely to develop GDV than dogs with broader chests. The overall mortality for surgically treated gastric dilatation-volvulus ranges from 10% to 18%, with most deaths occurring in patients that required splenectomy and partial gastrectomy.

Clinical signs of GDV include abdominal distention, unproductive vomiting or retching, lethargy, weakness, sometimes straining to defecate, and collapse. The owner may think that the animal is vomiting productively because of the white foamy froth (saliva) that is not able to pass into the twisted stomach. In some cases, there is a history of the dog's being fed a large meal or consuming a large quantity of water prior to the onset of clinical signs. Instruct the owner of any patient with a predisposition for and clinical signs of GDV to transport the animal to the nearest veterinary facility immediately.

Physical examination often reveals a distended abdomen with a tympanic area on auscultation. In dogs with very deep chests, it may be difficult to appreciate abdominal distention if the stomach is tucked up under the rib cage. Depending on the stage of shock,

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the patient may have sinus tachycardia with bounding pulses, cardiac dysrhythmias with pulse deficits, or bradycardia. The mucous membranes may appear red and injected or pale with a prolonged capillary refill time. The patient may appear anxious and attempt to retch unproductively. If the patient is nonambulatory at the time of presentation, the prognosis is more guarded.

The definitive diagnosis of GDV is based on clinical signs, physical examination findings, and radiographic appearance of gas distention of the gastric fundus with dorsocranial displacement of the pylorus and duodenum (the so-called “Double-Bubble” or “Popeye arm” sign) (Fig. 1-52). In simple gastric dilatation without volvulus, there is gas distention of the stomach with anatomy appearing normal on radiography. With “food bloat,” or gastric distention from overconsumption of food, ingesta is visible in the distended stomach (Fig. 1-53).

As soon as a patient presents with a possible GDV, place a large-bore intravenous catheter in the cephalic vein(s) and assess the patient’s ECG, blood pressure, heart rate, capillary refill time, and respiratory function. Obtain blood samples for a complete blood count, serum biochemistry profile, immediate lactate measurement, and coagulation tests *before* taking any radiographs. Rapidly infuse a colloid (hetastarch or Oxyglobin, 5 mL/kg IV bolus) along with shock volumes of a crystalloid fluid (up to 90 mL/kg/hour) (see section on Shock). Monitor perfusion parameters (heart rate, blood pressure, capillary refill time, and ECG) and titrate fluid therapy according to the patient’s response. The use of short-acting glucocorticosteroids is controversial. Glucocorticosteroids may help stabilize cellular membranes and decrease the mechanisms of ischemia-reperfusion injury, but no detailed studies have proved them to be beneficial versus not using glucocorticosteroids in the patient with GDV.

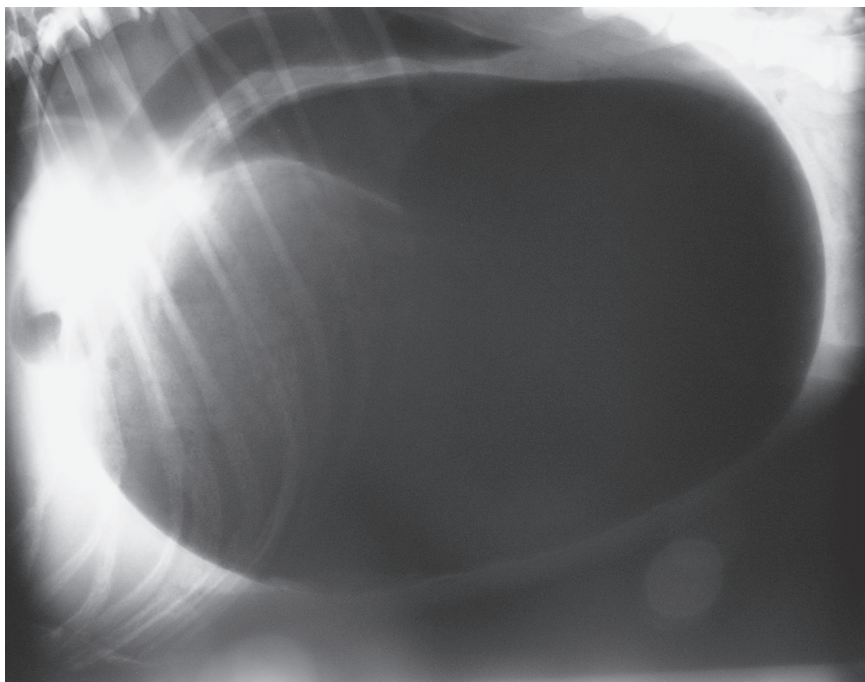


Figure 1-52: Example of gastric dilatation-volvulus (GDV), with characteristic dorsocranial displacement of the pylorus and proximal duodenum and gas distention of the gastric fundus. Always obtain a right lateral radiograph if the presence of GDV is suspected.

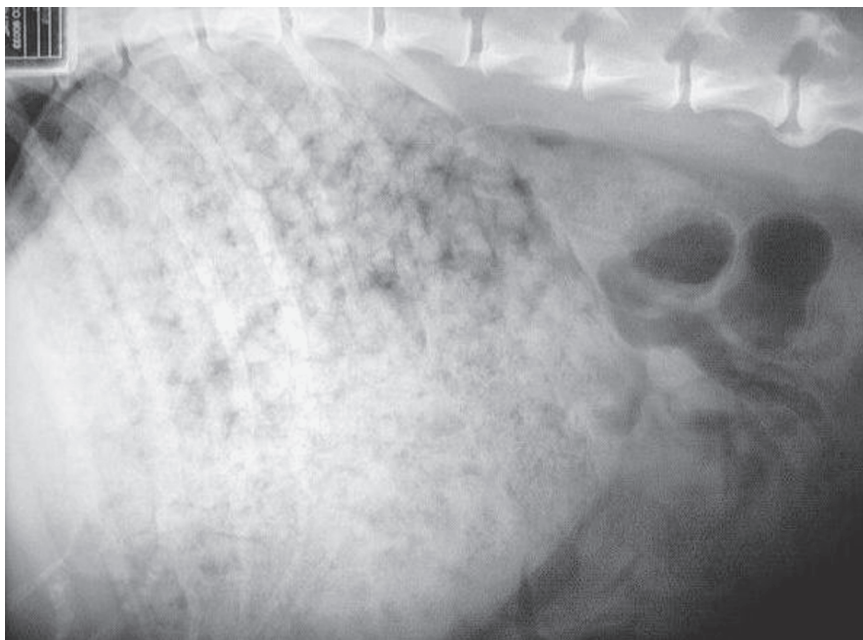


Figure 1-53: Example of “food bloat” with severe gastric distention caused by overconsumption of food. In rare cases, this can lead to decreased gastric perfusion, necrosis of the gastric wall, and perforation even without volvulus.

Attempt gastric decompression, either with placement of an orogastric tube or by trocharization. To place an orogastric tube, position the distal end of the tube at the level of the patient’s last rib (Fig. 1-54) and place it adjacent to the animal’s thorax; then put a piece of tape around the tube where it comes out of the mouth, once it is in place. Put a roll of 2-inch tape in the patient’s mouth behind the canine teeth and then secure the roll in place by taping the mouth closed around the roll of tape. Lubricate the tube with lubricating jelly and slowly insert the tube through the center of the roll of tape into the stomach. The passing of the tube does not rule out volvulus.

In some cases, the front legs of the patient need to be elevated, and the caudal aspect of the patient lowered (front legs standing on a table with back legs on the ground) to allow gravity to pull the stomach down to allow the tube to pass. Once the tube has been passed, air within the stomach is relieved, and the stomach can be lavaged. The presence of gastric mucosa or blood in the efflux from the tube makes the prognosis more guarded.

If an orogastric tube cannot be passed, clip and aseptically scrub the patient’s lateral abdomen and then insert 16-gauge over-the-needle catheter. “Pinging” the animal’s side with simultaneous auscultation allows determination of the location that is most tympanic—that is, the proper location for catheter insertion.

Once intravenous fluids have been started in the animal, take a *right lateral abdominal radiograph* to document GDV. If no volvulus is present, the owner may elect for more conservative care, and the animal should be monitored in the hospital for a minimum of 24 hours. Because some cases of GDV intermittently twist and untwist, the owner should be cautioned that although the stomach is not twisted at that moment, a volvulus can occur at any time. If radiographs demonstrate food bloat, induce emesis (apomorphine, 0.04 mg/kg IV) or perform orogastric lavage under general anesthesia. Documentation of gastric dilatation-volvulus constitutes a surgical emergency.

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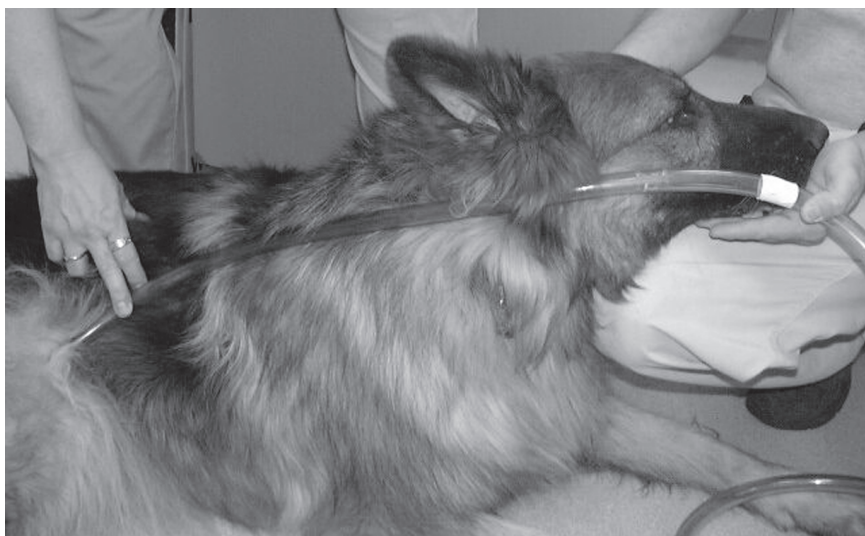


Figure 1-54: Measure the orogastric tube from the patient's mouth to the last rib and mark the tube to prevent pushing it in too far.

Following diagnosis of GDV, continue administration of intravenous fluids. Serum lactate measurements greater than 6.0 mmol/L are associated with an increased risk of gastric necrosis, requirement for partial gastrectomy, and increased mortality. Administer fresh frozen plasma (20 mL/kg) to patients with thrombocytopenia or prolonged PT, activated partial thromboplastin time (APTT), or activated clotting time (ACT). Cardiac dysrhythmias, particularly ventricular dysrhythmias, are common in cases of GDV and are thought to occur secondary to ischemia and proinflammatory cytokines released during volvulus and reperfusion. Lidocaine (1-2 mg/kg followed by 50 mcg/kg/minute IV CRI) can be used to treat cardiac dysrhythmias preemptively that are associated with ischemia-reperfusion injury, or administration can be started when ventricular dysrhythmias are present. Correct any electrolyte abnormalities, including hypokalemia and hypomagnesemia. The use of nonsteroidal antiinflammatory drugs (flunixin meglumine, carprofen, ketoprofen) that can potentially decrease renal perfusion and predispose to gastric ulcers is *absolutely contraindicated*. Administer analgesic drugs (fentanyl, 2 µg/kg IV bolus, followed by 3-20 µg/kg/hour IV CRI; or hydromorphone, 0.1 mg/kg IV) before anesthetic induction. After carrying out a balanced anesthesia protocol, the patient should be taken immediately to surgery for gastric derotation and gastropexy.

Postoperatively, assess the patient's ECG, blood pressure, platelet count, coagulation parameters, and gastric function (see section on Rule of Twenty). If no resection is required, the animal can be given small amounts of water beginning 12 hours after surgery. Depending on the severity of the patient's condition, small amounts of a bland diet can be offered 12 to 24 hours postoperatively. Continue supportive care with analgesia and crystalloid fluids until the patient is able to tolerate oral analgesic drugs (tramadol, 1-3 mg/kg PO q8-12h). Once the patient is ambulatory and able to eat and drink on its own, it can be released from the hospital; instruct the owner to feed the animal multiple small meals throughout the day for the first week.

SMALL INTESTINAL MESENTERIC VOLVULUS/TORSION

When the intestines twist around the root of the mesentery, a small intestinal or mesenteric volvulus occurs. The problem is most common in the young German Shepherd Dog,

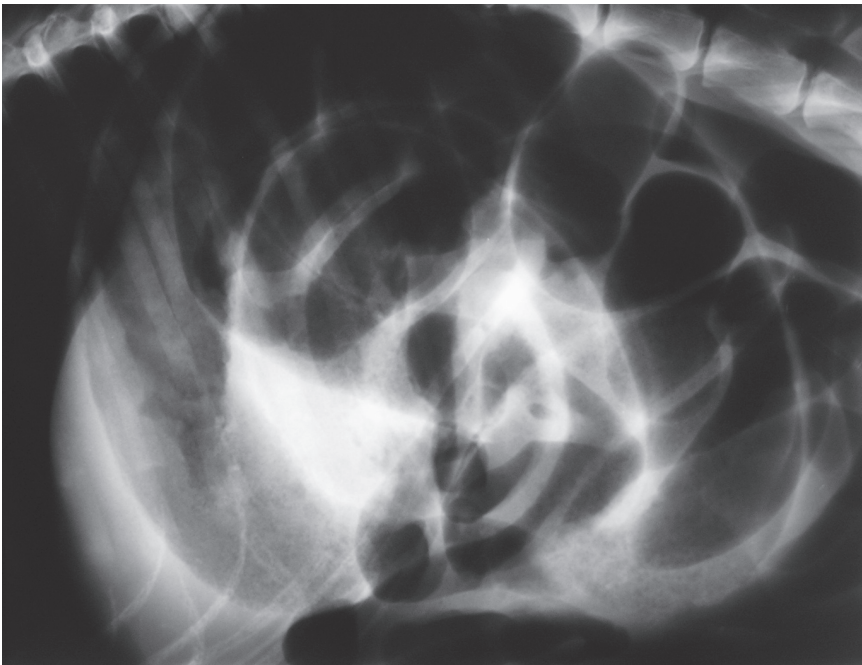


Figure 1-55: Severe generalized distention of the small intestine, characteristic of a mesenteric volvulus. This constitutes an immediate surgical emergency, and the prognosis is often poor. This condition is most common in young German Shepherd Dogs, but can be observed in any breed.

although it has been observed in other large and giant breeds. Predisposing factors include pancreatic atrophy, gastrointestinal disease, trauma, and splenectomy.

Clinical signs of mesenteric volvulus include vomiting, hemorrhagic diarrhea, bowel distention, acute onset of clinical signs of shock, abdominal pain, brick-red mucous membranes (septicemia), and sudden death.

Diagnosis is based on an index of suspicion and the presence of clinical signs in a predisposed breed. Plain radiographs often reveal grossly distended loops of bowel in a palisade gas pattern. In some dogs, multiple, tear-drop-shaped, gas-filled loops appear to rise from a focal point in the abdomen. Usually, massive distention of the entire small bowel is observed (Fig. 1-55). The presence of pneumoperitoneum or lack of abdominal detail secondary to the presence of abdominal fluid is characteristic of bowel perforation and peritonitis.

In a patient with mesenteric volvulus, immediate aggressive action is necessary for the animal to have any chance of survival. Treatment consists of massive volumes of IV crystalloid and colloid fluids (see section on IV Therapy), broad-spectrum antibiotics (ampicillin, 22 mg/kg IV qid, with enrofloxacin, 10 mg/kg IV once daily), and surgical correction of the bowel. Because of the massive release of proinflammatory cytokines, bacterial translocation, and ischemia, treatment for shock is of paramount importance (see sections on Rule of Twenty and Shock). Prognosis for any patient with mesenteric volvulus is poor.

LARGE INTESTINAL OBSTRUCTION

Obstipation

Obstipation (obstructive constipation) is most common in the older cat. In cases of simple constipation, rehydrating the animal with intravenous fluids and stool softeners is often

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sufficient for it to regain the ability to have a bowel movement. Obstipation, however, is caused by adynamic ileus of the large bowel that eventually leads to megacolon. Affected cats usually are anorectic, lethargic, and extremely dehydrated. Treatment consists of rehydration with intravenous crystalloid fluids, correction of electrolyte abnormalities, enemas, and promotility agents such as cisapride (0.5 mg/kg PO q8-24h). The use of phosphate enemas in cats is absolutely contraindicated because of the risk of causing acute, fatal hyperphosphatemia. In many cases, the patient should be placed under general anesthesia and manual deobstipation is performed with warm water soapy enemas and a gloved finger to relieve and disimpact the rectum. Stool softeners such as lactulose and docusate stool softer (DSS) may also be used. Predisposing causes of obstipation such as narrowing of the pelvic canal, perineal hernia, and tumors should be ruled out.

TUMORS OF THE GASTROINTESTINAL TRACT**Adenocarcinoma**

Adenocarcinoma is the most common neoplasm of the gastrointestinal tract that causes partial to complete obstruction. Adenocarcinomas tend to be annular and constricting, and they may cause progressive obstruction of the lumen of the small or large bowel. Siamese cats tend to have adenocarcinomas in the small intestine, whereas in dogs, the tumor tends to occur in the large intestine.

Clinical signs of adenocarcinoma are both acute and chronic and consist of anorexia, weight loss, and progressive vomiting that occur over weeks to months. Effusion may be present if metastasis to peritoneal surfaces has occurred.

Diagnosis is based on clinical signs and physical examination findings of a palpable abdominal mass, radiographic evidence of an abdominal mass and small or large intestinal obstruction, or ultrasonographic evidence of an intestinal mass.

Treatment consists of surgical resection of the affected bowel segment. The prognosis for long-term survival (10-12 months) is good if the mass is completely resected and if other clinical signs of cachexia or metastasis are observed at the time of diagnosis. Median survival is 15 to 30 weeks if metastasis to lymph nodes, liver, or the peritoneum are absent at the time of diagnosis. In dogs, the prognosis is more guarded.

Leiomyoma and Leiomyosarcoma

Leiomyoma and leiomyosarcoma are tumors that can cause partial or complete obstruction of the bowel. Clinical signs are often referred to progressive anemia, including weakness, lethargy, inappetence, and melena. Hypoglycemia can be observed as a paraneoplastic syndrome, or due to sepsis and peritonitis secondary to bowel perforation. Leiomyomas are most commonly observed at the ceco-colic junction or in the cecum. Surgical resection and anastomosis is usually curative, and has a favorable prognosis.

STRANGULATED HERNIAS

Incarceration of a loop of bowel into congenital or acquired defects in the body wall can cause small bowel obstruction. Pregnant females and young animals with congenital hernias are most at risk. Rarely, older animals with perineal hernias and animals of any age with traumatic hernias can be affected. Clinical signs are consistent with a small intestinal obstruction: anorexia, vomiting, lethargy, abdominal pain, and weakness. Diagnosis is often made based on physical examination of a reducible or nonreducible mass in the body wall. Hernias whose contents are reducible are usually asymptomatic. Treatment consists of supportive care and rehydration, administration of broad-spectrum antibiotics, and surgical correction of the body wall hernia. In some cases, intestinal resection and anastomosis of the affected area is necessary when bowel ischemia occurs.

BOWEL PERFORATION

The potential for bowel perforation should be suspected whenever there is any penetrating injury (knife, gunshot wound, bite wound, stick impalement) of the abdomen. Injuries that result in bowel ischemia and rupture can also occur secondary to nonpenetrating blunt

trauma or shear forces (e.g., big dog–little dog/cat). Perforation of the stomach and small and large intestines can occur with use of nonsteroidal antiinflammatory drugs.

Diagnosis of bowel perforation first depends on the alertness to the possibility that the bowel may have been perforated or penetrated. As a general rule, all penetrating injuries of the abdomen should be investigated by exploratory laparotomy. Diagnostic peritoneal lavage (DPL) can be performed; however, early after penetrating injury of the bowel, DPL may be negative or nondiagnostic until peritonitis develops. Whenever any patient with blunt or penetrating abdominal trauma does not respond to initial fluid therapy, or responds and then deteriorates, the index of suspicion for bowel injury should be raised. The findings of pneumoperitoneum on abdominal radiographs or of intracellular bacteria, extracellular bacteria, bile pigment, bowel contents, and cloudy appearance of fluid obtained by abdominocentesis or diagnostic peritoneal lavage fluid (see sections on Abdominocentesis and Diagnostic Peritoneal Lavage) warrant immediate surgical exploration.

Treatment largely consists of stabilizing the patient's cardiovascular and electrolyte status with intravenous fluids, administration of broad-spectrum antibiotics, and definitive surgical exploration and repair of injured structures.

RECTAL PROLAPSE

Prolapse of the rectum is observed most frequently secondary to parasitism and gastro-intestinal viral infections in young puppies and kittens with chronic diarrhea. Older animals with rectal prolapse often have an underlying problem such as a tumor or mucosal lesion that causes straining and dyschezia. The diagnosis of a rectal prolapse is made based on physical examination findings. The diagnosis of rectal prolapse is sometimes difficult to distinguish from small intestinal intussusception. In rare cases, the intussusception can invaginate through the large bowel, rectum, and anus. The two entities are distinguished from one another by inserting a lubricated thermometer or blunt probe into the cul-de-sac formed by the junction of the prolapsed mucosa and mucocutaneous junction at the anal ring. Inability to insert the probe or thermometer indicates that the rectal mucosa is prolapsed. Passage of the probe signifies that the prolapsed segment is actually the intussusceptum.

Treatment can be performed easily if the prolapse is acute and the rectal mucosa is not too irritated or edematous. The presence of severely necrotic tissue warrants surgical intervention. To reduce an acute rectal prolapse, after placing the patient under general anesthesia, lubricate the prolapsed tissue and gently push it back into the rectum, using a lubricated syringe or syringe casing. Apply a loose purse-string suture, leaving it in place for a minimum of 48 hours. De-worm the patient and administer stool softeners. If a rectal prolapse cannot be reduced, or if the tissue is nonviable, surgical intervention is warranted.

In patients in which viable tissue does not stay reduced with a purse-string suture, a colopexy can be performed during a laparotomy. First, place tension on the colon to reduce the prolapse, and then suture the colon to the peritoneum of the lateral abdominal wall with two to three rows of 2-0 or 3-0 monofilament suture material. If the prolapsed tissue is nonviable, it must be amputated. Place four stay sutures at 90-degree intervals through the wall of the prolapse at the mucocutaneous junction. Resect the prolapse distal to the stay sutures and then reestablish the rectal continuity by suturing the seromuscular layers together in one circumferential line and the mucosal layers together in the other. Replace the suture incision into the anal canal. Following surgery, de-worm the patient and administer a stool softener and analgesic drugs. Avoid using thermometers or other probes in the immediate postoperative period because they may disrupt suture lines.

ACUTE GASTRITIS

Acute gastritis may be associated with a variety of clinical conditions, including oral hemorrhage, ingestion of highly fermentable nondigestible foods or garbage, toxins, foreign bodies, renal or hepatic failure, inflammatory bowel disease, and bacterial and viral infections. Diarrhea often accompanies or follows acute gastritis. Hemorrhagic gastroenteritis often occurs as a shock-like syndrome with a rapidly rising hematocrit level. Clinical signs

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of gastritis include depression, lethargy, anterior abdominal pain, excessive water consumption, vomiting, and dehydration. Differential diagnosis of acute gastritis includes pancreatitis, hepatic or renal failure, gastrointestinal obstruction, and toxicities (Box 1-44).

The diagnosis is often a diagnosis of exclusion of other causes (see preceding text). A careful and thorough examination of the vomitus may be helpful in arriving at a diagnosis. A complete blood count, serum biochemistry profile including amylase and lipase, parvovirus test (in young puppies), fecal flotation and cytology, abdominal radiographs (plain and/or contrast studies), and abdominal ultrasound may be warranted to rule out other causes of acute vomiting.

While diagnostic tests are being performed, treatment consists of withholding all food and water for a minimum of 24 hours. After calculating the patient's degree of dehydration, administer a balanced crystalloid fluid to normalize acid-base and electrolyte status. Control vomiting with antiemetics such as metoclopramide, prochlorperazine, chlorpromazine, dolasetron, and ondansetron (Table 1-37). If vomiting is accompanied by diarrhea, administer broad-spectrum antibiotics (cefazolin, 22 mg/kg IV q8h, with metronidazole, 10 mg/kg IV q8h; or ampicillin, 22 mg/kg IV q6h, with enrofloxacin, 10 mg/kg IV q24h) to decrease the risk of bacterial translocation and bacteremia/septicemia. Although antacids (famotidine, ranitidine, cimetidine) do not have a direct antiemetic effect, their use can decrease gastric acidity and esophageal irritation during vomiting. If gastritis is secondary to uremia or nonsteroidal antiinflammatory drug use, administer gastroprotectant and antiemetic drugs (ranitidine, 1 mg/kg PO q12h; sucralfate, 0.25-1 g/dog PO q8h; or omeprazole (0.5-1 mg/kg PO Q24h) to decrease acid secretion and coat areas of gastric ulceration (Table 1-37). Once food and water can be tolerated, the patient can be placed on an oral diet and medications, and intravenous fluids can be discontinued.

BOX 1-44 CAUSES OF ACUTE GASTRITIS

- Bacterial toxin
- Brain lesion
- Dietary indiscretion
- Drugs
- Food allergy
- Hepatic failure
- Infectious disease
- Renal failure
- Stress
- Toxic chemicals
- Trauma

TABLE 1-37 Antiemetic Drugs and Dosages

Drugs (Proprietary name)	Suggested dosages*
Phenothiazines	
Chlorpromazine (Thorazine)	0.2-0.5 mg/kg IM q8h, 0.05 mg/kg IV q4h, 1.0 mg/kg per rectum q8h (dog)
Prochlorperazine (Compazine)	0.1 mg/kg IM q6h, 0.5 mg/kg IV, IM q8h
Serotonin antagonists	
Dolasetron (Anzemet)	0.1-0.3 mg/kg IV q24h
Ondansetron (Zofran)	0.6-1.0 mg/kg IV q12h
Others	
Metoclopramide (Reglan) [†]	0.2-0.5 mg/kg SQ q8h, 1.0-2.0 mg/kg/day IV, 3 mg/kg IM q8h (dog)

*All doses apply to dogs and cats unless otherwise noted.

[†]Do not use until a gastrointestinal obstruction has been ruled out.

HEMORRHAGIC GASTROENTERITIS

Hemorrhagic gastroenteritis (HGE) is an acute onset of severe hemorrhagic vomiting and diarrhea most commonly observed in young small-breed dogs (e.g., Poodles, Miniature Dachshunds, Miniature Schnauzers) 2 to 4 years of age. Clinical signs develop rapidly and include vomiting and fetid diarrhea with hemorrhage, often strawberry jam–like in appearance. The hematocrit can rise from 55% to 75%. Often, the animal is extremely hypovolemic but has no apparent signs of abdominal pain. There is no known cause of HGE, although *Clostridium perfringens*, *Escherichia coli*, *Campylobacter*, and viral infections have been suggested but not consistently confirmed. Other differential diagnoses of of hematemesis and hemorrhagic diarrhea include coronavirus, parvovirus, vascular stasis, sepsis, hepatic cirrhosis with portal hypertension, and other causes of severe shock.

Immediate treatment consists of placement of a large-bore intravenous catheter and replenishment of intravascular fluid volume with crystalloid fluids (up to 90 mL/kg/hour), while carefully monitoring the patient's hematocrit and total protein.

Administer broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6h, and enrofloxacin 10 mg/kg IV q24h) because of the high risk of bacterial translocation and sepsis. Control vomiting with antiemetic drugs. Monitor the patient's platelet count and coagulation tests for impending disseminated intravascular coagulation (DIC), and administer fresh frozen plasma and heparin, as needed (see section on Disseminated Intravascular Coagulation). When vomiting has ceased for 24 hours, offer the animal small amounts of water, and then a bland diet (e.g., boiled chicken and rice or boiled ground beef and rice mixed with low-fat cottage cheese).

PANCREATITIS

Pancreatitis occurs most frequently in dogs but can occur in cats as well. In dogs, the onset of pancreatitis is sometimes preceded by ingestion of a fatty meal or the administration of drugs (e.g., potassium bromide or glucocorticoids). Glucocorticoids can increase the viscosity of pancreatic secretions and induce ductal proliferation, resulting in narrowing and obstruction of the lumen of the pancreatic duct. Pancreatitis can also occur following blunt or penetrating abdominal trauma, high duodenal obstruction causing outflow obstruction of the pancreatic papilla, pancreatic ischemia, duodenal reflux, biliary disease, and hyperadrenocorticism.

In cats, acute necrotizing pancreatitis is associated with anorexia, lethargy, hyperglycemia, icterus, and sometimes acute death. Chronic pancreatitis is more common in cats and results in intermittent vomiting, anorexia, weight loss, and lethargy. Predisposing causes of chronic pancreatitis in cats include pancreatic flukes, viral infection, hepatic lipidos, drugs, organophosphate toxicity, and toxoplasmosis.

Clinical signs of acute pancreatitis include sudden severe vomiting, abdominal pain, and lethargy. Depending on the severity of pancreatic inflammation, depression, hypotension, and systemic inflammatory response syndrome (SIRS) may be present. Subacute cases may have minimal clinical signs. Severe pancreatic edema can result in vascular changes and ischemia that perpetuates severe inflammation. Hypovolemic shock and DIC can also decrease pancreatic perfusion. Severe pancreatic edema, autolysis, and ischemia lead to pancreatic necrosis. Duodenal irritation is manifested as both vomiting and diarrhea. Pain may be localized to the right upper abdominal quadrant or may be generalized if peripancreatic saponification occurs. Differential diagnosis of pancreatitis is the same as for any other cause of vomiting.

Complications that occur in patients with severe pancreatitis include dehydration, acid-base and electrolyte abnormalities, hyperlipemia, hypotension, and localized peritonitis. Hepatic necrosis, lipidos, congestion, and abnormal architecture can develop. Inflammatory mediators (bradykinin, phospholipase A, elastase, myocardial depressant factor, and bacterial endotoxins) stimulate the inflammatory cascade and can lead to SIRS, with severe hypotension, clotting system activation, and DIC. Electrolyte imbalances and hypovolemia secondary to vomiting all can lead to multiple organ dysfunction syndrome (MODS), and ultimately, death. If a patient survives an episode of acute pancreatitis, long-term sequelae

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can include diabetes mellitus. Monitor patients with recurrent pancreatitis for clinical signs of polyuria, polydipsia, polyphagia, hyperglycemia, and glucosuria.

The diagnosis of pancreatitis is based on the presence of clinical signs (which may be absent in cats), laboratory findings, and ultrasonographic evidence of pancreatic edema and increased peripancreatic echogenicity. Serum biochemistry analyses can sometimes support a diagnosis of pancreatitis; however, serum amylase and lipase are often unreliable indicators of pancreatitis, depending on the chronicity of the process in the individual patient. Both serum amylase and lipase are excreted in the urine. Impaired renal clearance/function can cause artifactual elevations of serum amylase and lipase in the absence of pancreatic inflammation. Furthermore, serum lipase levels can be elevated as a result of gastrointestinal obstruction (e.g., foreign body). Early in the course of the disease, levels can be two to six times normal, but they may decrease to within normal ranges at the time of presentation to the veterinarian. The transient nature of amylase elevation makes this test difficult to interpret, and it is not highly sensitive if a normal value is found. Lipase levels also increase later in the course of the disease. Amylase and lipase should be tested concurrently with the rest of the biochemistry profile.

Other changes often observed are elevations in BUN and creatinine levels secondary to dehydration and prerenal azotemia, hyperglycemia, and hyperlipemia. Hypocalcemia can occur secondary to peripancreatic fat saponification, and its presence warrants a more negative prognosis. A more specific measure is pancreatic lipase immunoreactivity, which becomes elevated in dogs and cats with pancreatitis. This test, combined with ultrasonographic or computed tomography evidence of pancreatitis, is the most sensitive and specific test available for making an accurate diagnosis. However, because the results of this test take time to obtain, animals must be treated in the meantime.

Abdominal effusion or fluid from diagnostic peritoneal lavage can be compared with serum amylase and lipase activity. Abdominal lipase and amylase concentrations in the fluid greater than that in the peripheral blood are characteristic of chemical peritonitis associated with pancreatitis. WBC counts greater than 1000 cells/mm³, the presence of bacteria, toxic neutrophils, glucose levels less than 50 mg/dL, or lactate levels greater than that of serum are characteristic of septic peritonitis, and immediate exploratory laparotomy is warranted. If a biopsy sample obtained during laparotomy does not demonstrate inflammation, but this does not rule out pancreatitis, because disease can be focal in nature and yet cause severe clinical signs.

Abdominal radiographs may sometimes reveal a loss of abdominal detail or a ground glass appearance in the right upper quadrant. Pancreatic edema and duodenal irritation can displace the gastric axis toward the left, toward the left with dorsomedial displacement of the proximal duodenum (the so-called “backwards 7” or “shepherd’s crook” sign). Ultrasonography and CT are more sensitive in making a diagnosis of pancreatitis.

Treatment of pancreatitis is largely supportive in nature and is designed to correct hypovolemia and electrolyte imbalances, prevent or reverse shock, maintain vital organ perfusion, alleviate discomfort and pain, and prevent vomiting (see section on Rule of Twenty). When treating pancreatitis in dogs, all food and water should be restricted. However, food should not be withheld from cats with chronic pancreatitis. Give fresh frozen plasma to replenish alpha-2-macroglobulins. Administer antiemetics such as chlorpromazine (use with caution in a hypovolemic or hypotensive patient), dolasetron, ondansetron, or metoclopramide to prevent or control vomiting. Analgesic drugs can be provided in the form of constant rate infusion (fentanyl, 3-7 µg/kg/hour IV CRI, and lidocaine, 30-50 µg/kg/minute IV CRI), intrapleural injection (lidocaine, 1-2 mg/kg q8h), or intermittent parenteral injections (morphine, 0.25-1 mg/kg SQ, IM; hydromorphone, 0.1 mg/kg IM or SQ). Because the pancreas must be rested, consider using parenteral nutrition.

ACUTE HEPATIC FAILURE

Acute hepatic failure may be associated with toxins, adverse reaction to prescription medication, and bacterial or viral infections. The most frequent clinical signs observed in a patient with acute hepatic failure are anorexia, lethargy, vomiting, icterus, bleeding, and

BOX 1-45 CAUSES OF ACUTE HEPATIC FAILURE**1****ENDOGENOUS HEPATOTOXINS**

Bacterial endotoxins

ENVIRONMENTAL TOXINS

Aflatoxin

Carbon tetrachloride

Dimethylnitrosamine

Heavy metals, herbicides

Pesticides

Phosphorus

Pyrrolizidine alkaloids

Selenium

EXOGENOUS DRUGS

Acetaminophen

Arsenicals

Azathioprine

Carprofen

Griseofulvin

Halothane

Ketoconazole

Mebendazole

Methoxyflurane

Phenazopyridine

Phenytoin

Sulfonamides (trimethoprim sulfadiazine, tetracycline)

INFECTIOUS AGENTS

Infectious canine hepatitis

Salmonella spp.*Leptospira* spp.

Feline infectious peritonitis virus

*Toxoplasma gondii**Bacillus piliformis* (Tyzzer's disease)**OTHERS**

Pancreatitis

Septicemia

Inflammatory bowel disease

Acute hemolytic anemia

CNS depression or seizures (associated with hepatic encephalopathy). Differential diagnosis and causes of acute hepatic failure are listed in Box 1-45.

Diagnosis of acute hepatic failure is based on clinical signs and biochemical evidence of hepatocellular (AST, ALT) and cholestatic (Alk Phos, T Bili, GGT) enzyme elevations. Ultrasonography may be helpful in distinguishing the architecture of the liver, but unless a mass or abscess is present, cannot provide a specific diagnosis of the cause of the hepatic damage.

Management of the patient with acute hepatic failure includes correction of dehydration and acid-base and electrolyte abnormalities, as shown in the following list:

- Hypoalbuminemia: Plasma or concentrated albumin. Plasma also is an excellent source of clotting factors that can become depleted.
- Clotting abnormalities: Vitamin K₁ (2.5 mg/kg SQ or PO q8-12h) to
- Severe anemia: Fresh or stored blood
- Gastric hemorrhage: Gastroprotectant drugs (omeprazole, ranitidine, famotidine, cimetidine, sucralfate)
- Hypoglycemia: Dextrose supplementation (2.5%-5%)
- Hepatic failure, particularly when hypoglycemia is present: Broad-spectrum antibiotics (ampicillin 22 mg/kg IV q6h; with enrofloxacin, 5 mg/kg IV q24h)
- Hepatic encephalopathy: lactulose or Betadine enemas
- Cerebral edema: Mannitol (0.5-1.0 g/kg IV over 10 to 15 minutes) followed by furosemide (1 mg/kg IV 20 minutes later). Deterioration of clinical signs may signify the development of cerebral edema.

Additional Reading

Applewhite AA, Cornell KK, Selcer BA: Diagnosis and treatment of intussusception in dogs. *Comp Cont Educ Pract Vet* 24(2):110-126, 2002.

Applewhite AA, Hawthorne JC, Cornell KK: Complications of enteroplication for the prevention of intussusception recurrence in dogs: 35 cases (1989-1999). *J Am Vet Med Assoc* 219(10):1415-1418, 2001.

Bertoy RW: Megacolon in the cat. *Vet Clin North Am Small Anim Pract* 32(4):901-915, 2002.

Coleman M, Robson M: Pancreatic masses following pancreatitis: pancreatic pseudocysts, necrosis and abscesses. *Comp Cont Educ Pract Vet* 27(2):147-154, 2005.

1

- Ferreri JA, Hardam E, Kimmel SE, et al: Clinical differentiation of acute necrotizing from chronic nonsuppurative pancreatitis in cats: 63 cases (1996-2001). *J Am Vet Med Assoc* 223(4):469-474, 2003.
- Holm JL, Chan DL, Rozanski EA: Acute pancreatitis in dogs. *J Vet Emerg Crit Care* 13(4):201-213, 2003.
- Junius G, Appeldoorn AM, Schrauwen E: Mesenteric volvulus in the dog: a retrospective study of 12 cases. *J Small Anim Pract* 45(2):104-107, 2004.
- Kemmel SE, Washabau RJ, Drobatz KJ: Incidence and prognostic value of low plasma ionized calcium concentration in cats with pancreatitis: 46 cases (1996-1998). *J Am Vet Med Assoc* 219(8):1105-1109, 2001.
- MacPhail C: Gastrointestinal obstruction. *Clin Tech Small Anim Pract* 17(4):78-183, 2002.
- Mansfield CS, Jones BR: Review of feline pancreatitis. Part 2: Clinical signs, diagnosis and treatment. *J Feline Med Surg* 3(3):125-132, 2001.
- Monnet E: Gastric dilatation-volvulus syndrome in dogs. *Vet Clin North Am Small Anim Pract* 33(5):987-1105, 2003.
- Ruax CG: Diagnostic approach to acute pancreatitis. *Clin Tech Small Anim Pract* 18(4):245-249, 2003.
- Ruax CG: Pathophysiology of organ failure in severe acute pancreatitis in dogs. *Comp Cont Educ Pract Vet* 22(6):531-542, 2000.
- Simpson KW: The emergence of feline pancreatitis. *J Vet Intern Med* 15(4):327, 2001.
- Steiner J: Diagnosis of pancreatitis. *Vet Clin North Am Small Anim Pract* 33(5):1181-1195, 2003.
- Washabau RJ: Gastrointestinal motility disorders and gastrointestinal prokinetic therapy. *Vet Clin North Am Small Anim Pract* 33(5):1007-1028, 2003.
- Watson P, Herrtage M: Chronic pancreatitis in dogs. *Vet Rec* 152(11):340, 2003.
- Watson PT: Exocrine pancreatic insufficiency as an end-stage of pancreatitis in 4 dogs. *J Small Anim Pract* 44(7):306-312, 2003.
- White RN: Surgical management of constipation. *J Feline Med Surg* 4(3):129-138, 2002.

HYPERTENSION: SYSTEMIC

Systemic hypertension is a recognized syndrome in dogs and cats and occurs most commonly secondary to acute or chronic renal failure, and less commonly as a primary idiopathic disease entity. Risk factors for the development of systemic hypertension in dogs and cats include renal insufficiency, hyperadrenocorticism, hyperthyroidism, pheochromocytoma, diabetes mellitus, polycythemia vera, hyperaldosteronism, hypertensive encephalopathy, acromegaly, intracranial hemorrhage, and CNS trauma.

Often, systemic hypertension is diagnosed when the animal is seen by the veterinarian because of some other clinical sign, such as acute blindness, retinal detachment, hyphema, epistaxis, and CNS signs following intracranial hemorrhage. Diagnosis of systemic hypertension is often difficult in the absence of clinical signs and without performing invasive or noninvasive blood pressure monitoring. Normal blood pressure (BP) measurements in dogs and cats are listed in Table 1-38.

Hypertension is defined as a consistent elevation in systolic BP >200 mm Hg, consistent diastolic BP >110 mm Hg, and consistent mean arterial blood pressure >130 mm Hg. The effects of systemic hypertension include left ventricular hypertrophy, cerebrovascular accident, renal vascular injury, optic nerve edema, hyphema, retinal vascular tortuosity, retinal hemorrhage, retinal detachment, vomiting, neurologic defects, coma, and excessive bleeding from cut surfaces.

TABLE 1-38 Normal Blood Pressure Measurements of Dogs and Cats

Species	Systolic (mm Hg)	Diastolic (mm Hg)	Mean (mm Hg)
Dog	100-160	80-120	90-120
Cat	120-150	70-130	100-150

TABLE 1-39 Drugs Used to Treat Systemic Hypertension

Drug	Canine dosage	Feline dosage
Angiotensin-converting enzyme inhibitors		
Enalapril	0.5-1.0 mg/kg PO q12-24h	0.25-0.5 mg/kg PO q12-24h
Benazepril	0.25-0.5 mg/kg PO q12-24h	Same as dog
α-Adrenergic blocker		
Prazosin	0.5-2.0 mg PO q12h	Not used
β-Adrenergic blockers		
Propranolol	2.5-10.0 mg PO q8-12h	2.5-5.0 mg PO q8-12h
Atenolol	0.25-1.0 mg/kg PO q12-24h	6.25-12.5 mg PO q12-24h
Calcium channel blockers		
Amlodipine	0.05-0.2 mg/kg PO q24h	0.625-1.25 mg PO q24h
Thiazide diuretics		
Hydrochlorothiazide	20.0-40.0 mg/kg PO q12-24h	
Loop diuretics		
Furosemide	2.0-4.0 mg/kg PO q12-24h	
Phthalazine derivatives		
Hydralazine	0.5-2.0 mg/kg PO q8-12h	2.5 mg PO q12-24h

Patients with systemic hypertension should have a thorough diagnostic work-up to determine the underlying cause. Although uncommon, hypertensive emergencies can occur with pheochromocytoma, acute renal failure, and acute glomerulonephritis. Sodium nitroprusside (1-10 μ /kg/minute IV CRI) or diltiazem (0.3-0.5 mg/kg IV given slowly over 10 minutes, followed by 15 μ /kg/minute) can be used to treat systemic hypertension. With the use of sodium nitroprusside or diltiazem, monitor carefully for hypotension.

Diagnosis is based on consistent elevations in systolic, diastolic, and/or mean arterial BP. Because many of the clinical signs associated with systemic hypertension involve hemorrhage into some closed cavity, other causes of hemorrhage, such as vasculitis, thrombocytopenia, thrombocytopathia, and hepatic or renal failure, should be investigated (see section on coagulation disorders). Diagnostic testing is based on clinical signs and index of suspicion for an underlying disease and may include a complete blood count; urinalysis; urine protein:creatinine ratio; ACTH stimulation test; thoracic and abdominal radiographs; thoracic and abdominal ultrasound; tick serology; brain CT or MRI; and assays of serum electrolytes, aldosterone concentration, T4, endogenous TSH, plasma catecholamine, and growth hormone.

Management of systemic hypertension involves treatment of the primary underlying disorder, whenever possible. Long-term adjunctive management includes sodium restriction in the form of cooked or prescription diets to decrease fluid retention. Obese animals should be placed on dietary restrictions and undergo a weight reduction program. Thiazide and loop diuretics may be used to decrease sodium retention and circulating blood volume. Alpha- and beta-adrenergic blockers may be used, but they are largely ineffective as monotherapeutic agents for treating hypertension. Calcium channel blockers and angiotensin-converting enzyme (ACE) inhibitors are the mainstay of therapy in the treatment of hypertension in dogs and cats (Table 1-39).

Additional Reading

- Acierno MJ, Labato MA: Hypertension in dogs and cats. *Comp Cont Educ Pract Vet* 26(5):336-346, 2004.
- Chastain CB, Panciera D, Elliot J, et al: Feline hypertension: clinical findings, and response to antihypertensive treatment in 30 cases. *J Am Anim Pract* 42(3):122-129, 2002.

- Cooke KL, Snyder PS: Diagnosing hypertension in dogs and cats. *Vet Med* 96(2):145-149, 2001.
- DeLaforcade AM, Rozanski EA: Central venous pressure and arterial blood pressure measurements. *Vet Clin North Am Small Anim Pract* 31(6):1163-1174, 2001.
- Littman MP, Fox PR: Systemic hypertension: recognition and treatment. In Fox PR, Sisson D, Moise NS (eds): *Textbook of canine and feline cardiology*. 2nd Edition. WB Saunders, Philadelphia, 1999.
- Selavka CM, Rozanski EA: Invasive blood pressure monitoring. In Wingfield WE (ed): *Veterinary emergency medicine secrets*. 2nd Edition. Hanley and Belfus, Philadelphia, 2000.

METABOLIC EMERGENCIES

DIABETIC KETOACIDOSIS

Diabetic ketoacidosis (DKA) is a potentially fatal and terminal consequence of unregulated insulin deficiency and possible glucagon excess. In the absence of insulin, unregulated lipolysis results in the beta-hydroxylation of fatty acids by abnormal hepatic metabolism. As a result, ketoacids—namely, acetoacetic acid, beta-hydroxybutyric acid, and acetone—are produced. Early in the course of the disease, patients exhibit clinical signs associated with diabetes mellitus: weight loss, polyuria, polyphagia, and polydipsia. Later, as ketoacids stimulate the chemoreceptor trigger zone, vomiting and dehydration occur, with resulting hypovolemia, hypotension, severe depression, abdominal pain, oliguria, and coma. At the time of presentation, often a strong odor of ketones (acetone) is present on the patient's breath.

Physical examination often reveals dehydration, severe depression or coma, and hypovolemic shock. In extreme cases, the patient exhibits a slow, deep Kussmaul respiratory pattern in an attempt to blow off excess CO_2 to compensate for the metabolic acidosis. A serum biochemistry profile and complete blood count often reveal prerenal azotemia, severe hyperglycemia (blood glucose >400 mg/dL), hyperosmolarity (>330 mOsm/kg), lipemia, hyponatremia (sodium >145 mEq/L), elevated hepatocellular and cholestatic enzyme activities, high anion gap, and metabolic acidosis. Although a whole body potassium deficit is usually present, the serum potassium may appear artifactually elevated in response to metabolic acidosis. With severe metabolic acidosis, potassium moves extracellularly in exchange for a hydrogen ion. Phosphorus too moves intracellularly in response to acidosis, and serum phosphorus is usually decreased. Hypophosphatemia >2 mg/dL can result in intravascular hemolysis. Urinalysis often reveals 4+ glucosuria, ketonuria, and a specific gravity of 1.030 or greater. The urine of all diabetic animals should be cultured to rule out a urinary tract infection or pyelonephritis.

Treatment of a patient with DKA presents a therapeutic challenge. Treatment is aimed at providing adequate insulin to normalize cellular glucose metabolism, correcting acid-base and electrolyte imbalances, rehydration and restoration of perfusion, correcting acidosis, providing carbohydrate sources for utilization during insulin administration, and identifying any precipitating cause of the DKA.

Obtain blood samples for a complete blood count, and serum biochemistry electrolyte profiles. Whenever possible, insert a central venous catheter for fluid infusion and procurement of repeat blood samples. Calculate the patient's dehydration deficit and maintenance fluid requirements and give appropriate fluid and electrolytes over a period of 24 hours. It is advisable to rehydrate patients with severe hyperosmolarity for a minimum of 6 hours before starting insulin administration. Use a balanced electrolyte solution (e.g., Plasmalyte-M, Normosol-R, lactated Ringer's solution) or 0.9% saline solution for maintenance and rehydration. Balanced electrolyte solutions contain small amounts of potassium and bicarbonate precursors that aid in the treatment of metabolic acidosis. Treat animals with severe metabolic acidosis with an $\text{HCO}_3^- >11$ mEq/L or a pH <7.1 with supplemental bicarbonate (0.25–0.5 mEq/kg). Add supplemental dextrose to the patient's fluids as a carbohydrate source during insulin infusion.

Both insulin and carbohydrates are necessary for the proper metabolism of ketone bodies in patients with DKA. The rate and type of fluid and amount of dextrose supplementation

will change according to the patient's blood glucose concentration. Serum potassium will drop rapidly as the metabolic acidosis is corrected with fluid and insulin administration. Measure serum potassium every 8 hours, if possible, and supplement accordingly (see section on Fluid Therapy for chart of potassium supplementation). If the patient's potassium requirement exceeds 100 mEq/L, or if the rate of potassium infusion approaches 0.5 mEq/kg/hour in the face of continued hypokalemia, magnesium should be supplemented. Magnesium is required as a cofactor for many enzymatic processes and for normal function of the Na,K-ATPase pump. Hypomagnesemia is a common electrolyte disturbance in many forms of critical illness. Replenishing magnesium (MgCl_2 , 0.75 mEq/kg/day IV CRI) often helps to correct the refractory hypokalemia observed in patients with DKA. Patients with hypophosphatemia that approaches 2.0 mmol/L should receive potassium phosphate (0.01-0.03 mmol/kg/hour IV CRI). When providing potassium phosphate supplementation, be aware of the additional potassium added to the patient's fluids, so as to not exceed recommended rates of potassium infusion. To determine the amount of potassium chloride (KCl) to add along with potassium phosphate (KPO_4), use the following formula:

$$\text{mEq K}^+ \text{ derived from KCl} = \text{Total mEq of K}^+ \text{ to be administered over 24 hours} - \text{mEq}$$

in which K^+ is derived from KPO_4

Clinical signs of severe hypophosphatemia include muscle weakness, rhabdomyolysis, intravascular hemolysis, and decreased cerebral function that can lead to depression, stupor, seizures, or coma.

Insulin administration

Regular insulin can be administered either IM or as a constant rate infusion in the treatment of patients with DKA. Subcutaneous insulin should not be administered. Because of the severe dehydration present in most patients with DKA, subcutaneous insulin is poorly absorbed and is not effective until hydration has been restored.

In the low-dose intravenous method, place regular insulin (1.1 units/kg for a cat, and 2.2 units/kg for a dog) in 250 mL of 0.9% saline solution. Run 50 mL of this mixture through the intravenous line to allow the insulin to adsorb to the plastic tubing. Administer the patient's insulin fluid rate according to blood glucose levels (Table 1-40). Adjust the patient's total fluid volume according to changes in the insulin fluid rate as necessary. In many cases, multiple bags of fluids are necessary because they must be changed when fluctuations in blood glucose concentrations occur in response to therapy. Infusion of the insulin mixture should be in a separate intravenous catheter. To replenish hydration, use a second intravenous line for the more rapid infusion of non-insulin-containing fluids.

To administer the regular insulin IM, first give 0.22 unit/kg IM and then re-check the patient's blood glucose every hour. Additional injections of regular insulin (0.11 unit/kg

TABLE 1-40 Type of Fluid and Rate of Insulin Infusion, Based on Patient's Blood Glucose Concentration, in the Treatment of Diabetic Ketoacidosis

Blood glucose (mg/dL)	Rate of insulin/0.9% NaCl infusion (mL/hour)	Other fluid type (mL/hour)
>250	10	0.9% NaCl
200-250	7	0.45% NaCl + 2.5% dextrose
150-200	5	0.45% NaCl + 2.5% dextrose
100-150	5	0.45% NaCl + 2.5% dextrose
<100	0	0.45% NaCl + 5% dextrose

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IM) should be administered based on the patient's response to subsequent injections. Once the patient's blood glucose falls to 200 to 250 mg/dL, add 2.5% to 5% dextrose to the fluids to maintain the blood glucose concentration at 200 to 300 mg/dL. Continue intramuscular injection of regular insulin (0.1-0.4 unit/kg q4-6h) until the patient is rehydrated, no longer vomiting, and able to tolerate oral fluids and food without vomiting. Even in patients with intramuscular regular insulin therapy, a central venous catheter should be placed for frequent blood sample collection. As the patient begins to respond to therapy, monitor electrolytes, glucose, and acid-base status carefully. Hypokalemia, hypophosphatemia, and hypomagnesemia can occur. When the patient's hydration and acid-base status has normalized and the patient is able to tolerate oral food and water, a longer-acting insulin can be administered as for treatment of a patient with uncomplicated diabetes.

HYPEROSMOLAR NONKETOTIC DIABETES

Extreme hyperosmolality can result in a coma, if uncorrected. In patients with diabetes mellitus, hyperglycemia and hypernatremia secondary to osmotic diuresis and free water loss can lead to severe hyperosmolality. In dogs, normal serum osmolality is <300 mOsm/L of serum. Hyperosmolality is expected when serum osmolality is >340 mOsm/L. If equipment for determining serum osmolality is not available, osmolality can be calculated by the following formula:

$$\text{Osm/L} = 2(\text{Na} + \text{K}) + (\text{glucose}/18) + (\text{BUN}/2.8)$$

Patients with severe dehydration, hyperglycemia, hypernatremia, and azotemia may experience cerebral edema without ketonemia. Treatment is directed solely at rehydrating the patient and slowly reducing blood glucose levels using a hypotonic solution such as 0.45% NaCl + 2.5% dextrose or 5% dextrose in water (D₅W). After the initial rehydration period, administer potassium supplementation conservatively.

HYPOLYCEMIA

Red blood cells and the brain absolutely depend on the oxidation of glucose for energy. Hypoglycemia can be caused by various systemic abnormalities that can be related to intestinal malabsorption of nutrients, impaired hepatic glycogenolysis or gluconeogenesis, and inadequate peripheral utilization of glucose. Clinical signs of hypoglycemia are extremely variable and can include weakness, tremors, nervousness, polyphagia, ataxia, tachycardia, muscle twitching, incoordination, visual disturbances, and generalized seizures. Clinical signs typically occur when serum glucose levels are <60 mg/dL. The combination of the clinical signs listed previously, documentation of low serum glucose, and alleviation of clinical signs upon glucose administration is known as *Whipple's triad*.

Whenever a patient presents with hypoglycemia, consider the following important factors: the age of onset, the nature of the hypoglycemic episode (transient, persistent, or recurrent), and the pattern based on the patient's history (Box 1-46).

Treatment of hypoglycemia is directed at providing glucose supplementation and determining any underlying cause. Administer supplemental dextrose (25%-50% dextrose, 2-5 mL/kg IV; or 10% dextrose, 20 mL/kg PO) as quickly as possible. Do not attempt oral glucose supplementation in any patient having a seizure or if the airway cannot be protected. Administer intravenous fluids (e.g., Normosol-R, lactated Ringer's solution, 0.9% saline solution) with 2.5%-5% supplemental dextrose until the patient is eating and able to maintain euglycemia without supplementation. In some cases (e.g., insulinoma), eating or administration of supplemental dextrose can promote insulin secretion and exacerbate clinical signs and hypoglycemia. In cases of refractory hypoglycemia secondary to iatrogenic insulin overdose, glucagon (50 mg/kg IV bolus, then 10-40 ng/kg/minute IV CRI) can also be administered along with supplemental dextrose. To make a glucagon infusion of 1000 ng/mL, reconstitute 1 mL (1 mg/mL) of glucagon according to the manufacturer's instructions and add this amount to 1000 mL of 0.9% saline solution.

BOX 1-46 CAUSES OF HYPOGLYCEMIA**ACCELERATED GLUCOSE REMOVAL**

Insulin overdose
 Ethanol poisoning
 Salicylate toxicity
 Propranolol
 Functional islet cell tumor
 Toxicity
 Oral hypoglycemic agents
 Renal glucosuria
 Hepatoma
 Endotoxemia

FAILURE OF GLUCOSE SECRETION

Functional hypoglycemia (nonrecognizable lesion)
 Neonatal hypoglycemia

“Toy breed hypoglycemia”
 “Hunting breed hypoglycemia”
 Starvation
 Hepatic enzyme insufficiencies
 Hypoadrenocorticism
 Hepatic insufficiency
 Malabsorption and starvation
 Large mesodermal tumors
 Sepsis
 Increased extrahepatic glucose substrate utilization
 Hematoma
 Hepatic abscess
 Renal failure
 Extrahepatic tumors

1**HYPOCALCEMIA: ECLAMPSIA (PUERPERAL TETANY)**

The diagnosis of eclampsia (puerperal tetany) is often made on the basis of history and clinical signs. Clinical signs can become evident when total calcium decreases to <8.0 mg/dL in dogs and <7.0 mg/dL in cats. The disease is often observed in small, excitable dogs, and stress may play a complicating role in the etiology. In most bitches, the disease manifests itself 1 to 3 weeks after parturition. In some cases, however, clinical signs can develop before parturition occurs. Hypophosphatemia may accompany hypocalcemia. Clinical signs of hypocalcemia include muscle tremors or fasciculations, panting, restlessness, aggression, hypersensitivity, disorientation, muscle cramping, hyperthermia, stiff gait, seizures, tachycardia, a prolonged QT interval on ECG, polydipsia, polyuria, and respiratory arrest.

Treatment of eclampsia consists of slow, cautious calcium supplementation (10% calcium gluconate, 0.15 mg/kg IV over 30 minutes). Severe refractory tetanus can be controlled with intravenous diazepam. Supportive care includes intravenous fluid administration and cooling (see section on Hyperthermia and Heat-induced Illness). Instruct the owner to give the patient oral calcium supplements (e.g., 1 to 2 tablets of Tums bid-tid) after discharge from the hospital. Also instruct the owner about how to wean the puppies, allowing the bitch to dry up, in order to prevent recurrence. Recurrence with subsequent pregnancies is common, particularly in patients that receive calcium supplementation during gestation (Table 1-41).

HYPERCALCEMIA

Hypercalcemia can occur from a variety of causes. The GOSH DARN IT mnemonic can be used to remember the various causes of hypercalcemia in small animal patients (Box 1-47).

The gastrointestinal, renal, and nervous systems are most commonly affected, particularly when serum total calcium rises above 16.0 mg/dL. Clinical signs of severe hypercalcemia include muscle weakness, vomiting, seizures, and coma. ECG abnormalities include prolonged PR interval, rapid QT interval, and ventricular fibrillation. The most serious clinical signs are often seen when hypercalcemia is observed in combination with hyperphosphatemia or hypokalemia. Pay special attention to the “calcium \times phosphorus product.” If this product exceeds 70, dystrophic calcification can occur, leading to renal failure. Renal complications include polyuria, polydipsia, dehydration, and loss of renal tubular concentrating ability. Renal blood flow and the glomerular filtration rate (GFR) are impaired when serum total calcium exceeds 20 mg/dL. The extent, location, and number of renal tubular injuries are the main factors in determining whether renal damage secondary to hypercalcemia is reversible or irreversible.

TABLE 1-41 Treatment of Hypocalcemia

Drug	Preparation	Available calcium	Dosage	Comments
<i>Parenteral calcium*</i>				
Calcium gluconate	10% solution	9.3 mg/mL	a. Slow IV to effect (0.5-1.5 mL/kg IV) b. 5-15 mg/kg/hr IV c. 1-2 mL/kg diluted 1:1 with saline SC t.i.d. 5-15 mg/kg/hr IV	Stop if bradycardia or shortened Q-T interval occurs; infusion to maintain normal Ca; may be given SC Only given IV, as extremely caustic perivascularly
Calcium chloride	10% solution	27.2 mg/mL		
<i>Oral calcium†</i>				
Calcium carbonate	Many sizes	40% tablet	25-50 mg/kg/day	Most common calcium supplement
Calcium lactate	325-, 650-mg tablets	13% tablet	25-50 mg/kg/day	
Calcium chloride	Powder	27.2%	25-50 mg/kg/day	May cause gastric irritation
Calcium gluconate	Many sizes	10%	25-50 mg/kg/day	
<i>Vitamin D</i>				
				Time for Maximal Effect to Occur
				Time for Toxic Effect to Resolve
Vitamin D ₃ (ergocalciferol)	Capsules, syrup, parenteral (IM)	–	<i>Initial:</i> 4000-6000 units/day <i>Maintenance:</i> 1000-2000 units/kg once daily to once weekly	5-21 days 1-18 wk
Dihydroxyvitamin D ₃ (1,25-dihydroxyvitamin D ₃)	Tablets, capsules, oral solution	–	<i>Initial:</i> 0.02-0.03 mg/kg/day <i>Maintenance:</i> 0.01-0.02 mg/kg q24-48h	1-7 days 1-3 wk
1,25-dihydroxyvitamin D ₃ (calcitriol)	Capsules	–	2.5 mg/kg/day	1-4 days 2-24 days

*Do not mix calcium solution with bicarbonate-containing fluids, as precipitation may occur.

†Calculate dose on elemental calcium content.

From DiBartola SP: Fluid Therapy in Small Animal Practice, Philadelphia, WB Saunders, 1992, p 169.

BOX 1-47 CAUSES OF HYPERCALCEMIA

- | | |
|--|---|
| <ul style="list-style-type: none"> • Granulomatous (fungal disease) • Osteogenic • Spurious (laboratory error) • Hyperparathyroidism • Vitamin D toxicosis • Addison's disease (hypoadrenocorticism) | <ul style="list-style-type: none"> • Renal failure • Neoplasia (lymphoma, multiple myeloma, osteosarcoma) • Idiopathic (cats) • Toxins and drugs (overzealous calcium administration; thiazide diuretics) |
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Emergency therapy of hypercalcemia is warranted when severe renal compromise, cardiac dysfunction, or neurologic abnormalities are present, or if no clinical signs occur but the calcium \times phosphorus product exceeds 70. The treatment of choice is correction of the underlying cause of hypercalcemia, whenever possible. In some cases, the results of diagnostic tests take time, and emergency therapy should be initiated immediately, before a definitive cause of the hypercalcemia is found. Emergency management of hypercalcemia consists of reduction of serum calcium levels. Administer intravenous fluids (0.9% saline solution) to expand extracellular fluid volume and promote calciuresis. To promote diuresis, initial intravenous fluid rates should approach two to three times maintenance levels (120–180 mL/kg/day). Potassium supplementation may be required to prevent iatrogenic hypokalemia. Administration of a loop diuretic such as furosemide (2–5 mg/kg IV) will promote calcium excretion. Calcitonin (4 IU/kg IM q12h for cats and 8 IU/kg IM q24h for dogs) can be administered to decrease serum calcium levels. In severe refractory hypercalcemia secondary to cholecalciferol toxicity, more aggressive calcitonin therapy (4–7 IU/kg SQ q6–8h) can be attempted. Side effects of calcitonin treatment include vomiting and diarrhea. Alternatively, bisphosphonates (pamidronate, 1.02–2.0 mg/kg IV) are useful in rapidly reducing serum calcium concentrations.

Glucocorticosteroids reduce calcium release from the bone, decrease intestinal absorption of calcium, and promote renal calcium excretion. Administer glucocorticosteroids only after the underlying cause of hypercalcemia has been determined and appropriate therapy started. Because many forms of neoplasia can result in hypercalcemia as a paraneoplastic syndrome, empiric use of glucocorticosteroids can induce multiple drug resistance, making the tumor refractory to the effects of chemotherapeutic agents.

ACUTE ADRENOCORTICAL INSUFFICIENCY

Hypoadrenocorticism is most commonly observed in young to middle-aged female dogs, but it can occur in animals of any age, gender, and breed. Clinical signs, which are referable to deficiency in glucocorticoid (cortisol) and mineralocorticoid (aldosterone) hormones, may develop slowly over time, leading to a waxing and waning course; acute clinical signs occur when >90% of the adrenal functional reserve has been destroyed. In such cases, complete adrenocortical collapse can result in an Addisonian crisis. Lack of aldosterone causes a lack of renal sodium and water retention, and impaired potassium excretion. The most significant clinical signs associated with hypoadrenocorticism are depression, lethargy, weakness, anorexia, shaking, shivering, vomiting, diarrhea, weight loss, abdominal pain, weakness, hypotension, dehydration, and inappropriate bradycardia (Box 1-48).

The diagnosis of hypoadrenocorticism is made based on the patient's clinical signs in combination with electrolyte abnormalities that include hyperkalemia, hyponatremia, and hypochloremia. Serum sodium concentration (115–130 mEq/L) is often greatly reduced, and serum potassium is elevated (>6.0 mEq/L). A sodium:potassium ratio of <27 is characteristic of hypoadrenocorticism, although not exactly pathognomonic. Electrocardiographic changes associated with hyperkalemia include inappropriate bradycardia, absence of p waves, elevated spiked T waves, and widened QRS complexes. Other more variable bloodwork abnormalities include a lack of a stress leukogram, eosinophilia, hypoglycemia, hyperphosphatemia, hypercalcemia, azotemia, and hypocholesterolemia. A definitive diagnosis of hypoadrenocorticism is based on an adrenocorticotrophic hormone (ACTH) stimulation test.

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BOX 1-48 BREED PREDISPOSITION TO HYPOADRENOCORTICISM

- Bassett Hound
- Bearded Collie
- Great Dane
- Great Pyrenees
- Portuguese Water Dog
- Standard Poodle
- West Highland White Terrier

In patients with hypoadrenocorticism, baseline cortisol levels are usually low, with a lack of appropriate cortisol release after administration of ACTH analogue. Rarely, animals with “atypical” hypoadrenocorticism lose glucocorticoid secreting ability from the zona fasciculata, but retain mineralocorticoid secretory ability from the zona glomerulosa. Atypical Addisonian patients have normal serum electrolytes but still have clinical signs of vomiting, diarrhea, weakness, lethargy, inappetence, muscle wasting, and weight loss. The diagnosis is more difficult in such cases because of the presence of normal electrolytes. An ACTH stimulation test should be considered, particularly in predisposed breeds.

Treatment of hypoadrenocorticism includes placement of a large-bore intravenous catheter, infusion of intravenous crystalloid fluids (0.9% saline solution), and replenishment of glucocorticoid and mineralocorticoid hormones. Administer dexamethasone or dexamethasone–sodium phosphate (0.5–1.0 mg/kg IV). Dexamethasone will not interfere with the ACTH stimulation test, unlike other longer-acting steroids (e.g., prednisolone, methylprednisolone sodium succinate, triamcinolone). Depending on the severity of the patient’s condition, consider monitoring using the Rule of Twenty. Administer antiemetics and gastroprotectant drugs to treat nausea, vomiting, and hematemesis. Give the patient broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6h) if hematochezia or hemorrhagic diarrhea is present. If severe gastrointestinal blood loss occurs, whole blood, packed red blood cells, or fresh frozen plasma may be required. Control hypoglycemia with 2.5%–5.0% dextrose. Use sodium bicarbonate, regular insulin with dextrose, or calcium gluconate to correct severe hyperkalemia with atrial standstill (see section on Atrial Standstill).

Chronic therapy for hypoadrenocorticism consists of mineralocorticoid and glucocorticosteroids supplementation for the rest of the animal’s life. Mineralocorticoid supplementation can be in the form of desoxycorticosterone pivalate (DOCP) (2.2 mg/kg IM) or fludrocortisone acetate (0.1 mg/2.5–5 kg body weight daily). Fludrocortisone acetate possesses both mineralocorticoid and glucocorticoid activities and can be used as the sole daily treatment of hypoadrenocorticism. (Because fludrocortisone is poorly absorbed in some dogs, it may not completely normalize electrolyte abnormalities in these animals.) DOCP is primarily a mineralocorticoid. Give supplemental glucocorticosteroids in the form of prednis(ol)one (1–0.25 mg/kg/day).

In dogs, iatrogenic hypoadrenocorticism can be caused by abrupt discontinuation of glucocorticosteroid treatment. Long-term glucocorticosteroid supplementation can down-regulate the pituitary gland’s excretion of endogenous ACTH and the zona fasciculata’s ability to excrete cortisol. However, the zona glomerulosa’s ability to secrete aldosterone does not appear to be affected. Clinical signs of iatrogenic hypoadrenocorticism include inability to compensate for stress, weakness, lethargy, vomiting, diarrhea, and collapse. Treatment of iatrogenic hypoadrenocorticism is the same as for naturally occurring disease. Following immediate emergency treatment, the patient should be weaned slowly from exogenous glucocorticosteroid supplementation.

THYROTOXICOSIS

Severe hyperthyroidism can manifest as a medical emergency as a result of hypermetabolism. Clinical signs in affected cats with severe thyrotoxicosis include fever, severe tachycardia (heart rate >240 bpm), vomiting, hypertension, congestive heart failure with pulmonary edema, and fulminant collapse. Clinical signs typically are manifested as an end-stage of chronic debilitation associated with hyperthyroidism and are often preceded by polyphagia, weight loss, cardiac murmur, polyuria/polydipsia (PU/PD), vomiting, and diarrhea.

Treatment of thyrotoxicosis includes antagonizing the adrenergic activity by administration of a beta-adrenergic blocker (esmolol, (25–50 μ /kg/minute, or propranolol, 0.02 mg/kg/hour). Administration of glucocorticosteroids (dexamethasone, 1 mg/kg) may inhibit the conversion of thyroxine (T_4) to the active form triiodothyronine (T_3) and decrease peripheral tissue responsiveness to T_3 , effectively blocking its effects. Correct hypoglycemia with supplemental dextrose (2.5%). Use care to avoid overhydration in a patient with cardiac failure or insufficiency. Start the patient on methimazole as quickly as possible and consider the use of radioactive iodine therapy.

Additional Reading

- Behrend EN: Clinical approach to hypercalcemia. *Vet Med* 97(10):763–769, 2002.
- Chastain CB, et al. Use of Pamidronate to reverse Vitamin D_3 –induced toxicosis in dogs. *Small Anim Clin Endocrinol* 10(2):10, 2000.
- Chastain CB, Panciera D, Waters C, et al: Glucagon constant rate infusion: a novel strategy for the management of hyperinsulinemic-hypoglycemic crisis in the dog. *Small Anim Clin Endocrinol* 10(3):18, 2000.
- Connally HE: Critical care monitoring considerations for the diabetic patient. *Clin Tech Small Anim Pract* 17(2):73–78, 2002.
- Drobatz KJ, Casey KK: Eclampsia in dogs: 31 cases (1995–1998). *J Am Vet Med Assoc* 217(2): 216–219, 2000.
- Fincham SC, Drobatz KJ, Gillespie TN, Hess RS: Evaluation of plasma ionized magnesium concentration in 122 dogs with diabetes mellitus: a retrospective study. *J Vet Intern Med* 18(5):615–617, 2004.
- Greco DS: Hypoadrenocorticism in dogs and cats. *Vet Med* 95(6):468–475, 2000.
- Hostutler RA, Chew DJ, Jaeger JQ, et al: Uses and effectiveness of pamidronate disodium for treatment of dogs and cats with hypercalcemia. *J Vet Intern Med* 19(1):29–33, 2005.
- Kerl ME: Diabetic ketoacidosis: pathophysiology and clinical and laboratory presentation. *Comp Cont Educ Pract Vet* 23(3):220–228, 2001.
- Kerl ME: Diabetic ketoacidosis: treatment recommendations. *Comp Cont Educ Pract Vet* 23(4):330–339, 2001.
- Koenig A, Drobatz KJ, Beale AB, King LG: Hyperglycemia, hyperosmolar syndrome in feline diabetics: 17 cases (1995–2001). *J Vet Emerg Crit Care* 14(1):30–40, 2004.
- Lathan P, Tyler J: Canine hypoadrenocorticism: pathogenesis and treatment. *Comp Cont Educ Pract Vet* 27(2):110–120, 121–133, 2003.
- Midkiff AM, Chew DJ, Randolph JF, et al.: Idiopathic hypercalcemia in cats. *J Vet Intern Med* 14(6):619–626, 2000.
- Morrow CK, Volmer PA: Hypercalcemia, hyperphosphatemia, and soft tissue mineralization. *Comp Cont Educ Pract Vet* 24(5):380–388, 2002.
- Vasilopoulos RJ, Mackin A: Humoral hypercalcemia of malignancy: diagnosis and treatment. *Comp Cont Educ Pract Vet* 25(2):128–136, 2003.

NEUROLOGIC EMERGENCIES

Four classes of neurologic injuries can seriously jeopardize a patient's life: head injuries, spinal cord and vertebral column injuries, coma, and seizure. The separate entities are discussed in this section.

HEAD INJURIES

Head injuries can be associated with skin and superficial lacerations, concussions, fractures, and hemorrhage (intracranial and extracranial). Fractures include extracranial, linear, and depressed intracranial. Hemorrhage can be extradural, intradural, subdural, subarachnoid, and intracerebral. Immediately perform a baseline physical examination of an animal with head trauma at the time of presentation to assess neurologic status and determine whether progressive deterioration exists (Table 1–42).

During the initial examination, note the patient's ABC's (airway, breathing, and circulation). If necessary, establish an airway. Always supply supplemental oxygen to maintain $SpO_2 > 90\%$. Place an intravenous catheter and start increments of a shock dose of intravenous fluids (1/4 of 90 mL/kg/hour for dogs; 44 mL/kg/hour for cats). In order

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TABLE 1-42 Localizing Signs in Patients Presenting with Head Trauma

Clinical sign	Description	Anatomic location
Decerebrate rigidity	Extensor rigidity of all four limbs	Caudal midbrain, pontine, or rostral opisthotonus Cerebellar
Decerebellate	Extensor rigidity of forelimbs, tucked or flexed hind limbs, opisthotonus	Caudal cerebellar
Tetraplegia	Plegia of all four limbs	Pontomedullary or cervical spine
Hemiplegia	Plegia of front and hind limbs on same side, opposite side unaffected	Ipsilateral pontomedullary or cervical spine
Hemiparesis	Paresis of front and hind limbs on same side, opposite side unaffected	Opposite rostral brainstem or cerebrum
Torticollis or head aversion	Neck torsion, turning head and neck to one side	Contralateral midbrain-pontine tegmental

BOX 1-49 LEVELS OF CONSCIOUSNESS

Alertness	Alert, responsive, appropriate reaction to external stimuli
Depression	Appears lethargic and has sluggish response to external stimuli
Confusion	May appear confused or aggressive
Delirium	Vocalization, inappropriate response to external stimuli
Semicoma	Unconscious but responds to external noxious stimuli
Coma	Unconscious with no response to noxious stimuli

to maintain cerebral perfusion pressure, blood pressure must be normalized. If other concurrent injuries are suspected (e.g., pulmonary contusions), administer synthetic colloid fluids (Dextran-70, 5-10 mL/kg IV, or hetastarch, 5-10 mL/kg IV) to normalize blood pressure. Although the use of colloids is controversial because of their potential to leak into the calvarium, the benefits of reestablishing cerebral perfusion far outweigh the risks of their use. Hypertonic saline (7.5% NaCl, 3-5 mL/kg IV) can also be administered over 10 to 15 minutes to expand intravascular volume. Maintain blood glucose within normal reference ranges whenever possible, because hyperglycemia is a negative prognostic indicator in cases of head trauma. If tremors or seizures cause hyperthermia or increased metabolism, active cooling of the patient is warranted (see sections on Hyperthermia and Heat-Induced Injury). All patients with head trauma should receive care and monitoring based on the Rule of Twenty (see section on Rule of Twenty).

THE EMERGENCY NEUROLOGIC EXAMINATION

Examine the patient's level of consciousness, response to various stimuli, pupil size and reactivity to light, physiologic nystagmus, and cranial nerve deficits. In dogs, damage to the midbrain often produces coma and decerebrate rigidity. Initial consciousness followed by a unconsciousness or stupor usually involves an injury to the brainstem. Brainstem lesions can be caused by compressive skull fractures, extradural or subdural hematomas, or herniation through the foramen magnum from cerebral edema (Box 1-49).

The patient's pupil size and response to light can be used to localize a diagnosis and give a rough prognosis for severity of disease and possibility for return to function. Pupils can be normal in size, mydriatic, or miotic. Whenever a pupil appears miotic, direct ocular

injury with uveitis or secondary miosis due to brachial plexus injury should be ruled out. The eyes should always be examined to rule out ocular trauma.

In a patient with head trauma, a change from dilated to constricted to normal pupil size is suggestive of improvement in clinical function. Bilateral mydriatic pupils that are unresponsive to light in an unconscious animal are a grave prognostic sign and usually indicate an irreversible severe midbrain contusion. Bilateral miotic pupils with normal nystagmus and ocular movements are associated with diffuse cerebral or diencephalic lesions. Miotic pupils that become mydriatic indicate a progressive midbrain lesion with a poor prognosis. Unilateral, slowly progressive pupillary abnormalities in the absence of direct ocular injury are characteristic of brainstem compression or herniation caused by progressive brain swelling. Asymmetric pupils are seen in patients with rostral brainstem lesions and can change rapidly. Unresponsive pupils that are seen in the midposition occur with brainstem lesions that extend into the medulla and are a grave sign.

Visual deficits are common with intracranial injury. Lesions that are less severe and limited to the cerebrum produce contralateral menace deficits with normal pupillary light response. Bilateral cerebral edema can cause blindness with a normal response to light if the midbrain is not disturbed. A patient that is severely depressed and recumbent may not respond to menacing gestures, even when visual pathways are intact. Ocular, optic tract, optic nerve, or optic chiasm lesions can interfere with vision and the pupillary light response. Brainstem contusion and cerebral edema may produce blindness and dilated unresponsive pupils due to disturbance of the oculomotor area.

Examine all cranial nerves carefully. Cranial nerve abnormalities can indicate direct contusion or laceration of the neurons in the brainstem or where they exit the skull. Cranial nerves that are initially normal then later lose function indicate a progressively expanding lesion. When specific cranial nerve deficits are present, the prognosis is considered guarded.

Clinical signs such as rolling to one side, torticollis, head tilt, and abnormal nystagmus are usually associated with petrosal bone or cerebellomedullary lesions that produce vestibular neuron dysfunction. Fractures of the petrosal temporal bone often cause hemorrhage and cerebrospinal fluid (CSF) leak from the external ear canal. If the lesion is limited to the membranous labyrinth, the loss of balance will be toward the injured side and the quick phase of the nystagmus will be toward the injured side.

Normal physiologic nystagmus requires that the pathway is between the peripheral vestibular neurons and the pontomedullary vestibular nuclei to the nuclei of the cranial nerves that innervate the extraocular muscles (III, IV, VI). Severe brainstem lesions disrupt this pathway. Disruption of the pathway is manifested as an inability to produce normal physiologic nystagmus by moving the patient's head from side to side. In patients with severe central nervous system depression, this reflex may not be observed.

Next, assess postural changes and motor function abilities. A loss of the normal oculocephalic ("dolls-eye") reflex is an early sign of brainstem hemorrhage and a late sign of brainstem compression and herniation.

Any intracranial injury may be accompanied by a concurrent cervical spinal cord injury. Handle animals with such injuries with extreme care to avoid causing further damage. Whenever there is uncertainty whether a spinal cord lesion exists, strap the patient down to a flat surface and obtain radiographs of the spine. At least two orthogonal views may be required to see fractures; however, do not manipulate the patient until radiography has been completed. Crosstable views, in which the Bucky is turned perpendicular to the patient's spine, with a radiograph plate secured behind the patient, may be required to minimize patient motion. In patients with cerebral lesions, hemiparesis usually resolves within 1 to 3 days.

Evaluation of cranial nerve function at frequent intervals may reveal an initial injury or a progressively expanding lesion in the brain. Signs of vestibular disorientation, marked head tilt, and abnormal nystagmus occur with contusions of the membranous labyrinth and fracture of the petrous temporal bone. Hemorrhage and cerebrospinal fluid otorrhea may be visible from the external ear canal. Rolling movements indicate an injury to the cerebellar-medullary vestibular system.

1

Respiratory dysfunction and abnormal respiratory patterns are sometimes observed with severe head injury. Lesions of the diencephalon produce Cheyne-Stokes respirations, in which the patient takes progressively larger and larger breaths, pauses, then takes progressively smaller and smaller breaths. Mesencephalic lesions cause hyperventilation and can result in respiratory alkalosis. Medullary lesions result in a choppy, irregular respiratory pattern. Clinical signs of respiratory dysfunction in the absence of primary respiratory damage indicate a guarded prognosis.

After injury, seizures may be associated with intracranial hemorrhage, trauma, or an expanding intracranial mass lesion. Immediately begin medical therapy to control the seizure. Administer diazepam (0.5 mg/kg IV or 0.1-0.5 mg/kg/hour IV CRI) to treat seizures. If diazepam is not effective in combination with other treatments to control intracranial edema, consider giving pentobarbital (3-25 mg/kg IV to effect). Loading doses of phenobarbital (16-20 mg/kg IV divided into 4 or 5 doses, given every 20 to 30 minutes) may be beneficial in preventing further seizures.

Severe refractory seizures or decreased mentation may be associated with cerebral edema and increased intracranial pressure. Mannitol, an osmotic diuretic, is effective at reducing cerebral edema (0.5-1.0 g/kg IV over 10 to 15 minutes). Mannitol also acts as a free radical scavenger that can inhibit the effects of cerebral ischemia-reperfusion injury. Mannitol works synergistically with furosemide (1 mg/kg IV given 20 minutes after the mannitol infusion). Corticosteroids have not been demonstrated to be beneficial in the treatment of head trauma and may induce hyperglycemia. Hyperglycemia has been shown to be a negative prognostic indicator in cases of head trauma. Also, glucocorticoids can suppress immune system function and impair wound healing. Because of the known risks and lack of known benefits of glucocorticosteroids, their use in treatment of head trauma is contraindicated.

The prognosis for any patient with severe head trauma is guarded. Management of head trauma patients may include intense nursing care for a period of weeks to months, depending on the presence and extent of concurrent injuries. If progressive loss of consciousness occurs, surgery for decompression of compressive skull injuries should be considered.

The most common injury associated with head trauma in small animals is a contusion with hemorrhage in the midbrain and pons. Subdural or extradural hemorrhage with space-occupying blood clots is uncommon. Diagnostic tests of head trauma may include skull radiographs, CT, and MRI of the brain. Special studies can help detect edema and hemorrhage in the brain and brainstem, and aid in making an accurate diagnosis and prognosis. A cerebrospinal fluid tap is contraindicated in patients with head trauma because of the risk of causing a rapid decrease in intracranial pressure and brainstem herniation. If a compressive skull fracture is present, the patient should be stabilized for surgery to remove the compression. Surgery to alleviate increased intracranial pressure is rarely performed in veterinary medicine because of the poor prognosis and results. In some cases, when a lesion can be localized to one area, 1- to 2-cm burr holes can be placed through the skull over the affected area of the cerebrum, exposing the underlying brain tissue. Blood clots can be removed through the holes. The bone flap may or may not be replaced, depending on the surgeon's preference and the degree of brain swelling.

SPINAL CORD INJURIES

Spinal cord injuries may be associated with trauma, disk rupture, fractures, and dislocation of the spinal column. Proceed with caution when moving a patient with suspected spinal cord injury. Avoid flexion, extension, and torsion of the vertebral column. All animals that are unconscious following a traumatic event should be considered to have cervical or thoracolumbar spinal injury until proved otherwise by radiography, CT, or MRI. The animal should be moved onto a flat surface (e.g., board, door, window, picture frame) and taped down to prevent motion and further displacement of vertebrae. Sedation with analgesics or tranquilizers may be necessary to keep the animal immobile and to minimize patient motion. Whenever possible, avoid the use of narcotics in patients with head trauma because of the risk of increasing intracranial pressure. As in other emergencies, the ABCs

should be evaluated, and the patient treated for shock, hemorrhage, and respiratory compromise. Once the cardiovascular and respiratory systems have been evaluated and stabilized, a more thorough neurologic examination can be performed.

Thoracolumbar disease: herniated disks and trauma

Protrusion of an intervertebral disk indicates that the disk is bulging into the vertebral canal as a result of dorsal shifting of the nuclear pulposus disk material. Disk extrusion refers to the rupture of the outer disk membrane and extrusion of the nuclear material into the vertebral column. In dogs and cats, there are 36 intervertebral disks that potentially can cause a problem. Chondrodystrophic breeds of dogs are predisposed to endochondral ossification and include the Dachshund, Shih Tzu, French Bulldog, Bassett Hound, Welsh Corgis, American Spaniel, Beagle, Lhasa Apso, and Pekingese.

Initial examination of the patient with suspected intervertebral disk disease includes identifying the neuroanatomic location of the lesion based on clinical signs and neurologic deficits and then establishing a prognosis. The neurologic examination should be carried out without excessive manipulation of the animal. The presence of pain, edema, hemorrhage, or a visible deformity may localize an area of vertebral injury. Once an area of suspected lesion is localized based on physical examination findings, take radiographs to establish a diagnosis and to institute therapy. In most cases, the animal must receive a short-acting anesthetic for proper radiographic technique and to prevent further injury. Lateral and crosstable ventrodorsal (VD) or dorsoventral (DV) radiographs require less manipulation of the animal compared with traditional VD and DV projections. Myelography is often required to delineate the location of the herniated disk material.

Prognosis in spinal cord injury depends on the extent of the injury and the reversibility of the damage. Perception of noxious stimuli, or the presence of “deep pain,” by the animal when the stimulus is applied caudal to the level of the lesion is a good sign. To apply a noxious stimulus, apply firm pressure to a toe on one of the rear limbs using a thick hemostat or a pair of pliers. Flexion or withdrawal of the limb is simply a local spinal reflex, and should not be perceived as a positive response to or patient perception of the noxious stimulus. Turning of the head, vocalization, dilation of the pupils, change in respiratory rate or character, or attempts to bite are behaviors that are more consistent with perception of the noxious stimulus. Absence of perception of the noxious stimulus (“loss of deep pain”) is a very poor prognosis for return to function.

Focal lesions are usually associated with vertebral fractures and displacement of the vertebral canal. Focal lesions in one or more of the spinal cord segments from T₃ to T₄ can cause complete dysfunction of the injured tissue as a result of concussion, contusion, or laceration. The degree of structural damage cannot be determined from the neurologic signs alone. Transverse focal lesions result in paraplegia, with intact pelvic limb spinal reflexes and analgesia of the limbs and body caudal to the lesion. Clinical signs in patients with spinal injury are summarized in Table 1-43.

Treatment of spinal cord injuries

Carefully evaluate the cardiovascular and respiratory status of patients with spinal injuries. Immediately address specific injuries such as pneumothorax, pulmonary contusions, hypovolemic shock, and open wounds. If there is palpable or radiographic evidence of a vertebral lesion causing compressive injury, surgery is the treatment of choice unless the displacement has compromised most or all of the vertebral canal. Displacements through 50% to 100% of the vertebral canal are associated with a poor prognosis, particularly if deep pain is absent caudal to the lesion. In the absence of a radiographic lesion and in the presence of continued neurologic deficits, an MRI or CT scan or myelography is warranted to localize a potentially correctable lesion. Surgical exploration can be considered: with the objectives of providing spinal cord decompression by hemilaminectomy or laminectomy with removal of disk material or blood clots, realign and stabilize the vertebral column, and perform a meningoctomy, if necessary. Place the patient on a backboard or other rigid surface, taped down for transport and sedated, to be transported to a surgical specialist.

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TABLE 1-43 Localizing Signs in Patients with Spinal Trauma/Injuries

Location of lesion	Postural and reflex change
Cranial to C6	Spastic tetraplegia or tetraparesis Hyperreflexive all four limbs Severe injury can result in death from respiratory failure.
C6-T2	Tetraparesis or tetraplegia Depressed thoracic limb spinal reflexes (lower motor neuron) Hyperreflexive pelvic limbs (upper motor neuron)
T1-T3	Horner' syndrome (prolapsed nictitans, enophthalmos, and miosis)
T3-L3	Schiff-Sherrington syndrome (extensor rigidity of thoracic limbs, flaccid paralysis with atonia, areflexia, and analgesia of pelvic limbs)

BOX 1-50 CORTICOSTEROID DOSAGE IN ACUTE SPINAL TRAUMA*

Prednisolone sodium succinate or methylprednisolone, 20 to 30 mg/kg IV once, then 10 to 15 mg/kg IV at 3, 6, and 9 hours

*Potentially useful for injuries less than 8 hours old.
Adapted from Shores A: Spinal trauma, Vet Clin North Am Small Anim Pract 22:859, 1992.

The presence of worsening or ascending clinical signs may signify ascending-descending myelomalacia and is characteristic of a very poor prognosis. In acute spinal trauma, the use of glucocorticoids has been the mainstay of therapy; however, controversy exists about whether they actually offer any benefit. Traditional glucocorticosteroid therapy is listed in Box 1-50. More recently, the use of propylene glycol has proved to be beneficial in the treatment of acute traumatic herniated disk. High-dose glucocorticoids should only be used for the first 48 hours after initial injury. Side effects of glucocorticosteroid therapy include gastric and intestinal ulceration. The prophylactic use of gastroprotectant drugs will not prevent gastrointestinal ulcer formation; however, if signs of gastrointestinal ulcer are present, institute gastroprotectant therapy.

Management

Management of the patient with spinal cord injury includes aggressive nursing care and physical therapy. Many patients with spinal cord injury have little to no control over bladder function, which results in chronic dribbling or retention of urine and overdistention of the urinary bladder with overflow incontinence. Urinary bladder retention can lead to urinary tract infection, bladder atony, and overflow incontinence. Manual expression of the bladder several times a day may be enough to keep the bladder empty. Alternatively, place a urinary catheter to maintain patient cleanliness and to keep the bladder decompressed. (see section 5 on Urinary Catheterization).

Paralytic ileus and fecal retention are frequent complications of spinal cord injury. To help prevent constipation, provide highly digestible foods and maintain the patient's hydration with oral and intravenous fluids. Mild enemas or stool softeners can also be used to treat fecal retention. To prevent decubital ulcer formation, turn the patient every 4 to 6 hours, and use clean, dry, soft padded bedding. Apply deep muscle massage and passive range of motion exercises to prevent disuse atrophy of the muscles and dependent edema.

INJURIES TO THE PERIPHERAL NERVOUS SYSTEM

The radial nerve innervates the extensor muscles of the elbow, carpus, and digits. The radial nerve also supplies sensory innervation to the distal craniolateral surface of the forearm and the dorsal surface of the forepaw. Injuries to the radial nerve at the level of the elbow

result in an inability to extend the carpus and digits. As a result, the animal walks and bears weight on the dorsal surface of the paw. There is also loss of cutaneous sensation, which leads to paw injury. Injuries to the radial nerve above the elbow (in the shoulder area) results in an inability to extend the elbow and bear weight on the affected limb. It can take weeks before the full extent of the injury and any return to function are manifested. The animal may need to be placed in a carpal flexion sling or have eventual amputation if distal limb injury or self-mutilation occurs.

BRACHIAL PLEXUS

Sciatic nerve

The sciatic nerve primarily innervates the caudal thigh muscles that flex the stifle and extend the hip. The tibial branch of the sciatic nerve innervates the caudal leg muscles that extend the tarsus and flex the digits. The tibial nerve provides the sole cutaneous sensory innervation to the plantar aspect of the paw and digits. The peroneal branch of the sciatic nerve provides the sole sensory cutaneous innervation to the dorsal surface of the paw (Table 1-44). Sciatic nerve injury may occur with pelvic fractures, particularly those that involve the body of the ileum at the greater ischiatic notch, or with sacroiliac luxations that contuse the L6 and L7 spinal nerves that pass ventral to the sacrum to contribute to the sciatic nerve. With sciatic nerve injury, there is decreased stifle flexion and overflexion of the hock (tibial nerve), and the animal walks on the dorsal surface of the paw (peroneal nerve). Clinical signs of tibial or peroneal damage are seen with femur fractures or with inadvertent injection of drugs into the caudal thigh muscles.

Femoral nerve

The femoral nerve innervates the extensor muscles of the stifle. The saphenous branch of the femoral nerve provides the sole cutaneous innervation to an area on the medial distal thigh, the leg, and the paw. The femoral nerve is protected by muscles and is rarely injured in pelvic fractures. Clinical signs of femoral nerve injury are inability to support weight on the pelvic limb, absence of a patellar reflex, and analgesia in the area of cutaneous innervation.

COMA

Coma is complete loss of consciousness, with no response to noxious stimuli. In some animals that present in a coma or stuporous state, the immediate cause will be apparent. In other cases, however, a careful and thorough diagnostic work-up must be performed. A coma scale devised to assist in the clinical evaluation of the comatose patient is shown in Table 1-45. Whenever an animal presents in a comatose state, immediately secure the

TABLE 1 - 44 Localizing Signs of Patients with Forelimb Injury/Trauma

Location of injury	Clinical signs
C6-T2 nerve roots	Radial nerve paralysis
Musculocutaneous nerve	Inability to flex the elbow
Axillary or thoracodorsal	Dropped elbow nerve
Median and ulnar nerves	Loss of cutaneous sensation on the caudal surface of the forearm and palmar and lateral surfaces of the paw; inability to flex the carpus and digits
C8-T1 nerve roots	Radial, median, or ulnar nerve injury
C6-C7 nerve roots	Musculocutaneous, suprascapular, and axillary injury
C7-T3	Horner's syndrome (miosis, enophthalmos, and prolapsed nictitans)

TABLE 1-45 Small Animal Coma Scale (SACS)*

Motor activity	6
Normal gait, normal spinal reflexes	
Hemiparesis, tetraparesis, or decerebrate activity	5
Recumbent, intermittent extensor rigidity	4
Recumbent, constant extensor rigidity	3
Recumbent, constant extensor rigidity with opisthotonus	2
Recumbent, hypotonia of muscles, depressed or absent spinal reflexes	1
Brainstem reflexes	
Normal papillary reflexes and oculocephalic reflexes	6
Slow pupillary light reflexes and normal to reduced oculocephalic reflexes	5
Bilateral unresponsive miosis with normal to reduced oculocephalic reflexes	4
Pinpoint pupils with reduced to absent oculocephalic reflexes	3
Unilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	2
Bilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	1
Level of consciousness	
Occasional periods of alertness and responsive to environment	6
Depression of delirium, capable of responding to environment but response may be inappropriate	5
Semicomatose, responsive to visual stimuli	4
Semicomatose, responsive to auditory stimuli	3
Semicomatose, responsive only to repeated noxious stimuli	2
Comatose, unresponsive to repeated noxious stimuli	1

*Neurologic function is assessed for each of the three categories and a grade of 1 to 6 is assigned according to the descriptions for each grade. The total score is the sum of the three category scores. This scale is designed to assist the clinician in evaluating the neurologic status of the craniocerebral trauma patient. As a guideline and according to clinical impressions, a consistent total score of 3 to 8 represents a grave prognosis, 9 to 14 a poor to guarded prognosis, and 15 to 18 a good prognosis. (Modified from the Glasgow Coma Scale used in humans.)

From Shores A: Craniocerebral trauma. In Kirk RW, ed: Current Veterinary Therapy X. Small Animal Practice. Philadelphia, WB Saunders, 1989, p 849.

airway by placing an endotracheal tube (see section on Endotracheal Intubation). If necessary, provide respiratory assistance, or at a minimum, supplemental oxygen. Control existing hemorrhage and treat shock, if present.

Take a careful and thorough history from the owner. Make careful note of any seizure, trauma, or toxin exposure, and whether prior episodes of coma have ever occurred. Perform a careful physical examination, taking note of the patient's temperature, pulse, and respiration. An elevated temperature may suggest the presence of systemic infection, such as pneumonia or hepatitis, or a brain lesion with loss of hypothalamic thermoregulatory control. Very high temperatures associated with shock and coma are often observed in animals with heat stroke (see section on Heat Stroke and Heat-Induced Illness). Circulatory collapse or barbiturate overdose can produce coma and hypothermia.

Abnormal respiratory patterns also may be observed in a comatose patient. Hypoventilation may occur with elevated intracranial pressure or barbiturate overdose. Rapid respiratory rate may be associated with pneumonia, metabolic acidosis (DKA, uremia), or brainstem injury.

Examine the skin for any bruises or external trauma. Examine the mucous membranes and make note of color and capillary refill time. Icterus with petechiae or ecchymotic hemorrhage in a comatose patient may be associated with end-stage hepatic failure and hepatic encephalopathy. Smell the patient's breath for the odor of ketones that may signify DKA or end-stage hepatic failure.

Finally, conduct a complete neurologic evaluation. The presence of asymmetric neurologic signs may suggest an intracranial mass lesion (e.g., hemorrhage, neoplasia, injury). Usually, toxicities or metabolic disturbances (e.g., DKA, hepatic encephalopathy) cause symmetric clinical signs of neurologic dysfunction, with cerebral signs predominating. In hepatic encephalopathy, pupils are usually normal in size and responsive to light. In toxicities, the pupils are abnormal in size and may be unresponsive to light.

Obtain a complete blood count, serum biochemistry profile, urinalysis, and specific tests for glucosuria and ketonuria. Findings of a drastically elevated blood glucose with glucosuria, ketonuria, and high specific gravity are characteristic of DKA. Fever and uremic encephalopathy are characterized by severe azotemia with a low urine specific gravity. If barbiturate intoxication is suspected, save urine for later toxin analysis. Evaluate urine sediment for calcium oxalate crystalluria that may indicate ethylene glycol toxicity. Calculate plasma osmolality (see following section) to check for nonketotic hyperosmolar diabetes mellitus. Elevated blood ammonia levels may be associated with hepatic encephalopathy.

Diabetic Coma

In uncontrolled diabetes mellitus, hyperosmolality can result in clinical signs of disorientation, prostration, and coma. Plasma osmolality can be calculated from the formula:

$$\text{mOsm/L} = 2(\text{Na} + \text{K}) + (\text{glucose}/18) + (\text{BUN}/2.8)$$

Clinical signs of hyperosmolality can occur when the plasma osmolality exceeds 340 mOsm/L. Treatment of DKA or nonketotic hyperosmolar syndrome is aimed at reducing ketoacid production, stimulating carbohydrate utilization, and impeding peripheral release of fatty acids. The treatment of choice is rehydration and provision of supplemental regular insulin and a carbohydrate source (see section on Diabetic Ketoacidosis). During ketosis, insulin resistance may be present. Slow rehydration with 0.9% saline solution or other balanced crystalloid fluids (e.g., Normosol-R, Plasmalyte-M, lactated Ringer's solution), should occur, with the goal of rehydration over 24 to 48 hours. Too rapid rehydration can result in cerebral edema and exacerbation of clinical signs.

Hepatic Coma

Hepatic encephalopathy (HE) is characterized by an abnormal mental state associated with severe hepatic insufficiency. The most common cause of HE is congenital or acquired

TABLE 1-46

Grade of hepatic encephalopathy	Clinical signs
1	Listlessness, depression, mental dullness Personality changes Polyuria
2	Ataxia Disorientation Compulsive pacing or circling, head-pressing Apparent blindness Personality changes Salivation
3	Polyuria Stupor Severe salivation Seizures
4	Coma

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portosystemic shunts. Acute hepatic destruction can also be caused by toxins, drugs, or infectious causes. The treatment of HE is considered a medical emergency (Table 1-46). Absorption of ammonia and other nitrogenous substances from the gastrointestinal tract is thought to be one of the complicating factors in HE. Prevent absorption of ammonia and other nitrogenous substances from the gastrointestinal tract by restricting dietary protein to 15% to 20% for dogs, and to 30% to 35% (on a dry matter basis) for cats. Dietary protein should be from a nonanimal plant source (e.g., soybean) whenever possible. Caloric requirements are met with lipids and carbohydrates. Also prescribe cleansing enemas to rid the colon of residual material, and antibiotic therapy to reduce gastrointestinal tract bacteria. Neomycin (15 mg/kg q6h) can be administered as a retention enema. Metronidazole (7.5 mg/kg PO, q8-12h) or amoxicillin-clavulanate (16.25 mg PO q12h) can also be administered. Administer lactulose (2.5-5.0 mL q8h for cats; 2.5-15 mL q8h for dogs) to trap ammonia in the colon to prevent absorption (Table 1-46). Administer lactulose orally to an alert animal, or as a retention enema to a comatose animal. If lactulose is not available, Betadine retention enemas will change colonic pH and prevent ammonia absorption. A side effect of lactulose administration (PO) is soft to diarrheic stool.

EMERGENCY TREATMENT OF SEIZURES

A seizure is a transient disturbance of brain function that is sudden in onset, ceases spontaneously, and has a tendency to recur, depending on the cause. Most seizures are generalized and result in a loss of consciousness and severe involuntary contraction of the skeletal muscles, resulting in tonic-clonic limb activity and opisthotonus. Mastication, salivation, urination, and defecation are common. Partial (petit mal) seizures range from limited limb activity, facial muscle twitching, and episodic behavioral abnormalities to brief loss of consciousness. Similar clinical signs also can occur with syncopal episodes. Conduct a careful cardiac examination in any patient with a history of petit mal seizures. Seizures of any form constitute a medical emergency, particularly when they occur in clusters, or as *status epilepticus*.

Most seizures are of short duration and may have subsided by the time the animal is presented for treatment. Whenever a seizure occurs, however, it is important that the animal does not inadvertently injure itself or a bystander. It is important to evaluate whether the patient has a coexisting disease that can predispose it to seizures, such as hepatic failure, uremia, diabetes mellitus, hypoglycemia, toxin exposure, insulin-secreting tumors, and thiamine deficiency. Many toxins are responsible for clinical signs of tremors or seizures (see section on Poisons and Toxins). Treatment of a primary disease entity can help control seizures, in some cases, provided that the underlying cause is investigated and treated.

Status epilepticus, a state of continuous uncontrolled seizure activity, is a medical emergency. When an animal is in a state of status epilepticus, immediately place a lateral or medial saphenous intravenous catheter and administer diazepam (0.5 mg/kg IV) to help control the seizure. In most cases, the seizure must be controlled before a diagnostic work-up is attempted. Whenever possible, however, blood samples should be collected before administration of any anticonvulsant agent because of the risk of incorrect test results. For example, the propylene glycol carrier in diazepam can cause a false-positive ethylene glycol test using an in-house testing kit.

Whenever possible, check blood glucose levels, particularly in young puppies or kittens, to evaluate and treat hypoglycemia as a cause of seizures. If hypoglycemia exists, administer 25% dextrose (1 g/kg IV). If diazepam partially controls the status epilepticus, administer a constant rate infusion (0.1 mg/kg/hour in 5% dextrose in water). Diazepam is sensitive to light, and the bag and infusion line must be covered to prevent degradation of the drug. If diazepam fails to control status epilepticus, give pentobarbital (3-25 mg/kg IV to effect). The animal's airway should be intubated and protected while the patient is kept in the drug-induced coma. Protracted cases of seizures may require mannitol and furosemide therapy to treat cerebral edema.

Administer intravenous fluids (balanced crystalloid at maintenance doses [see section on Intravenous Fluid Therapy]). The patient should be turned every 4 to 6 hours to

prevent atelectasis. Insert a urinary catheter for cleanliness, and place the animal on soft dry padded bedding to prevent decubital ulcer formation. Depending on the length of time that the patient is rendered unconscious, apply passive range of motion exercises and deep muscle massage to prevent disuse atrophy of the muscles and dependent or disuse edema. Monitor the patient's oxygenation and ventilation status by arterial blood gas measurement or pulse oximetry and capnometry (see Section 5 on Blood Gas, Pulse Oximetry, and Capnometry). Administer supplemental oxygen to any patient that is hypoxemic secondary to hypoventilation or other causes. Severe refractory seizures can result in the development of neurogenic pulmonary edema. Lubricate the animal's eyes every 4 hours to prevent drying out and corneal abrasions. Depending on the cause of the seizure, administer phenobarbital at a loading dose of 16 to 20 mg/kg IV given in four to five injections, every 20 to 30 minutes; make sure that the patient is rousable in between injections).

Seizures in cats often are associated with structural brain disease. The occurrence of partial focal seizures is unequivocally associated with a focal cerebral lesion and acquired structural brain disease. An initial high frequency of seizures is also a strong indication that structural brain disease is present. Seizure activity in cats may occur as mild generalized seizures or complex partial seizures and may be associated with systemic disorders such as feline infectious peritonitis virus, toxoplasmosis, *Cryptococcus* infection, lymphosarcoma, meningiomas, ischemic encephalopathy, and thiamine deficiency.

Thiamine deficiency in the cat can be a medical emergency characterized by dilated pupils, ataxic gait, cerebellar tremor, abnormal oculocephalic reflex, and seizures. Treatment consists of administration of thiamine (50 mg/day) for three days.

Additional Reading

- Barnes HL, Chrisman CL, Mariani CL, Sims M, Alleman AR: Clinical signs, underlying cause, and outcome in cats with seizures: 17 cases (1997-2002). *J Am Vet Med Assoc* 225(11):1723-1726, 2004.
- Gandini G, Cizinauskas S, Lang J, et al: Fibrocartilaginous embolism in 75 dogs: clinical findings and factors influencing the recovery rate. *J Small Anim Pract* 44(2):76-80, 2003.
- Gordon PN, Dunphy ED, Mann FA: A traumatic emergency: handling patients with head injuries. *Vet Med* 98(9):788-798, 2003.
- Johnson J, Murtaugh R: Cranio cerebral trauma. In Bonagura J (ed): *Kirk's current veterinary therapy XIII*. Philadelphia, WB Saunders, 2000.
- Knipe ME, Vernau KM, Hornof WJ, LeCouteur RA: Intervertebral disc extrusion in six cats. *J Feline Med Surg* 3(3):161-168, 2001.
- Kraus K: Medical management of acute spinal cord disease. In Bonagura J, editor: *Kirk's current veterinary therapy XIII*. WB Saunders, Philadelphia, 2000.
- Mayhew PD, McLear RC, Ziemer LS, et al: Risk factors for recurrence of clinical signs associated with thoracolumbar intervertebral disk herniation in dogs: 229 cases (1994-2000). *J Am Vet Med Assoc* 225(8):1231-1236, 2004.
- Munana KR, Olby NJ, Sharp NJ, Skeen TM: Intervertebral disk disease in 10 cats. *J Am Anim Hosp Assoc* 37(4):384-389, 2001.
- Olby N, Levine J, Harris T, et al: Long-term functional outcome of dogs with severe injuries of the thoracolumbar spinal cord: 87 cases (1996-2001). *J Am Vet Med Assoc* 222(6):762-769, 2003.
- Platt SR: Feline seizure control. *J Am Anim Hosp Assoc* 37(6):515-517, 2001.
- Platt SR, Haag M: Canine status epilepticus: a retrospective study of 50 cases *J Small Anim Pract* 43(4):151-153, 2002.
- Saito M, Munana KR, Sharp NJ, Olby NJ: Risk factors for development of status epilepticus in dogs with idiopathic epilepsy and effects of status epilepticus on outcome and survival time: 32 cases (1990-1996). *J Am Vet Med Assoc* 219(5):618-623, 2001.
- Sammur V: Skills Laboratory Part I: Performing a neurologic examination. *Vet Med* 100(2):118-132, 2005.
- Sammur V: Skills Laboratory Part II: Interpreting the results of the neurologic examination. *Vet Med* 100(2):136-142, 2005.
- Somerville ME, Anderson SM, Gill PJ, et al: Accuracy of localization of cervical intervertebral disk extrusion or protrusion using survey radiography in dogs. *J Am Anim Hosp Assoc* 37(6):563-572, 2001.

1

Steffen F, Grasmueck S: Propofol for treatment of refractory seizures in dogs and a cat with intracranial disorders. *J Small Anim Pract* 41(11):496-499, 2000.

Syring RS: Assessment and treatment of central nervous system abnormalities in the emergency patient. *Vet Clin North Am Small Anim Pract* 35:343-358, 2005.

Syring RS, Otto CM, Drobatz KJ: Hyperglycemia in dogs and cats with head trauma: 122 cases (1997-1999). *J Am Vet Med Assoc* 218(7):1124-1129, 2001.

OCULAR EMERGENCIES

An ocular emergency is any serious condition that causes or threatens to cause severe pain, deformity, or loss of vision. Treat ocular emergencies immediately, within 1 to several hours after the emergency, whenever possible (Box 1-51, 1-52).

To assess the location and degree of ocular injury, perform a complete ocular examination. In some cases, short-acting sedation or general anesthesia in conjunction with topical local anesthetic may be necessary to perform the examination, because of patient discomfort and blepharospasm. The equipment listed in Box 1-53 may be necessary and may be invaluable in making an accurate diagnosis.

To perform a systematic and thorough ocular examination, first obtain a history from the owner. Has there been any prior incident of ocular disease? Is there any history of trauma or known chemical irritant or exposure? Did the owner attempt any irrigation or medical techniques prior to presentation? When was the problem first noticed? Has it changed at all since the owner noticed the problem?

After a history has been obtained, examine the patient's eyes for discharge, blepharospasm, or photophobia. If any discharge is present, note its color and consistency.

BOX 1-51 OCULAR EMERGENCIES REQUIRING IMMEDIATE THERAPY

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| <ul style="list-style-type: none">• Penetrating injury to the globe• Proptosis of the globe• Glaucoma• Corneal laceration• Acute corneal abrasion or ulcer• Acute iritis | <ul style="list-style-type: none">• Lid laceration• Descemetocoele• Orbital cellulitis• Chemical burns• Ocular foreign bodies• Hyphema |
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BOX 1-52 OCULAR EMERGENCIES THAT CAN CAUSE A SUDDEN LOSS OF VISION

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| <ul style="list-style-type: none">• Hyphema• Traumatic lid swelling• Exposure keratitis• Sudden acquired retinal degeneration• Retinal hemorrhage• Retinal detachment• Intracranial damage | <ul style="list-style-type: none">• Vitreous hemorrhage• Corneal edema• Acute glaucoma• Retinal detachment• Retinal edema• Traumatic avulsion of the optic nerve• Proptosis of the globe |
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BOX 1-53 EQUIPMENT NEEDED TO PERFORM AN OCULAR EXAMINATION

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| <ul style="list-style-type: none">• Loupe• Direct ophthalmoscope• Fine-tooth forceps• Lacrimal probe• Fluorescein sterile strips• Proparacaine (0.5%)• Short-acting mydriatics (tropicamide 1%) | <ul style="list-style-type: none">• Monocular indirect ophthalmoscope• Transilluminator• Lid retractor• Sterile saline eye wash in irrigation bottle• Sterile cotton-tipped swabs• Schiottz tonometer or Tonopen |
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Do not attempt to force the eyelids open if the patient is in extreme discomfort. Administer a short-acting sedative and topical local anesthetic such as 0.5% proparacaine. Note the position of the globe within its orbit. If the eye is exophthalmic, strabismus and protrusion of the third eyelid are often visible. Exposure keratitis may be present. In cases of retrobulbar or zygomatic salivary gland inflammation, the patient will resist opening the mouth and exhibit signs of discomfort or pain. Note any swelling, contusions, abrasions, or lacerations of the eyelids. Note whether the lids are able to close completely and cover the cornea. If a laceration of the lid is present, determine the depth of the laceration. Palpate the orbit for fractures, swelling, pain, crepitus, and cellulitis.

Examine the cornea and sclera for penetrating injury or foreign material. The use of lid retractors or small forceps can be very helpful in these cases. If a wound appears to penetrate completely into the globe, look for loss of uveal tissue, lens, or vitreous. Do NOT put any pressure on the globe, because intraocular herniation may result. Examine the conjunctiva for hemorrhage, chemosis, lacerations, and foreign bodies. Examine the superior and inferior conjunctival cul-de-sacs for foreign material. In such cases, placement of a topical anesthetic and use of a moistened cotton swab is invaluable to sweep the conjunctival fornix to pick up foreign bodies. Use a small, fine-tipped forceps to retract the third eyelid away from the globe and examine behind the third eyelid for foreign bodies.

Next, examine the cornea for opacities, ulcers, foreign bodies, abrasions, or lacerations. Place a small amount of fluorescein stain mixed with sterile water or saline on the dorsal sclera. Close the eye to disperse the stain over the surface of the cornea, then flush gently with sterile saline irrigation. Examine the cornea again for any defects. A linear defect perpendicular to the long axis of the eye should alert the clinician to investigate the conjunctiva for dystechia.

Record the pupil size, shape, and response to light (both direct and consensual). Examine the anterior chamber and note its depth and whether hyphema or aqueous flare are present. Is the lens clear and is it in the normal position? Lens luxation can cause the lens tissue to touch the cornea and cause acute corneal edema. Measure intraocular pressure with a Schiotz tonometer or Tonopen. Finally, dilate the pupil and examine the posterior chamber using a direct or indirect ophthalmoscope to look for intraocular hemorrhage, retinal hemorrhage, retinal detachment, tortuous retinal vessels, optic neuritis, and inflammation.

SPECIFIC CONDITIONS AND TREATMENT

The basic surgical instruments listed in Box 1-54 may be useful in the treatment of ocular lacerations and other ophthalmic injuries:

Injuries of the eyelids

Lid laceration

Bite wounds and automobile trauma commonly cause lacerations and abrasions of the lid margins. The lids can be considered to be two-layer structures, with the anterior composed of the skin and orbicularis muscle and the posterior layer composed of the tarsus and conjunctiva. The openings of the meibomian glands in the lid margin form the approximate line separating the lids into anterior and posterior segments. Splitting the lid into

BOX 1-54 BASIC INSTRUMENTS FOR TREATMENT OF OCULAR EMERGENCIES

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| <ul style="list-style-type: none"> • Castroviejo or Barraquer lid speculum • Bishop-Harmon tissue forceps • Stevens tenotomy scissors • Castroviejo corneal scissors • Castroviejo needle holder; standard jaws with lock • Beaver knife handle and No. 64 blades | <ul style="list-style-type: none"> • Lacrimal cannula, straight 22 gauge • Barraquer iris repository • Foreign body spud • Enucleation scissors, medium curve • Suture material: 6-0 silk, 4-0 nylon, 7-0 collagen, 6-0 ophthalmic gut, 7-0 nylon |
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these two segments facilitates the use of sliding skin flaps to close wound defects, if necessary.

Clean and thoroughly but gently irrigate the wound with sterile saline solution before attempting any lid laceration repair. Use sterile saline solution to irrigate the wound and conjunctiva. A 1% povidone-iodine scrub can be used on the skin, taking care to avoid getting any scrub material in the soft tissues of the eye. Drape the eye with an adhesive ocular drape, if possible, to prevent further wound contamination.

Trim the ragged wound edges, but be very conservative with tissue debridement. Leave as much tissue as possible to insure proper wound contracture with minimal lid deformity. Close a small lid wound with a figure-of-eight or two-layered simple interrupted suture of absorbable suture material or nylon in the skin. The lid margins must be absolutely apposed to prevent postoperative lid notching.

Ecchymosis of the lids

Direct blunt trauma to the eye can cause severe ecchymosis because of the excellent vascular supply of the eyelids. Other associated ocular injuries such as orbital hemorrhage, proptosis, and corneal laceration may also occur. Trauma, allergic reactions, inflammation of the sebaceous glands (hordeolum), thrombocytopenia, and vitamin K antagonist rodenticide intoxication can all cause ecchymoses of the lids.

Treat eyelid ecchymoses initially with cool compresses, followed by warm compresses. Resorption of blood can occur from 3 to 10 days after the initial insult. Ocular allergies respond well to topical application (dexamethasone ophthalmic ointment q6-8h) and systemic administration of glucocorticosteroids, along with cool compresses.

Conjunctival lacerations

In order to fully assess the conjunctiva for abnormalities, it may be necessary to carefully dissect it away from the underlying sclera. When performing this dissection, do not place undue pressure on the globe because of the risk of herniation of the intraocular contents through a scleral wound.

Repair large conjunctival lacerations with 6-0 absorbable sutures, using an interrupted or continuous pattern. Carefully approximate the margins of the conjunctiva to prevent formation of inclusion cysts. When large areas of the conjunctiva have been damaged, advancement flaps may be required to close the defect.

Subconjunctival hemorrhage

Subconjunctival hemorrhage is a common sequela of head trauma, and it may also be observed in various coagulopathies. By itself, it is not a serious problem but may signify severe underlying intraocular damage. A complete ocular examination is indicated. Other causes of subconjunctival hemorrhage include thrombocytopenia, autoimmune hemolytic anemia, hemophilia, leptospirosis, vitamin K antagonist rodenticide intoxication, severe systemic infection or inflammation, and prolonged labor (dystocia). Uncomplicated subconjunctival hemorrhage usually clears on its own within 14 days. If the conjunctiva is exposed because of swelling and hemorrhage, administer a topical protective triple antibiotic ophthalmic ointment every 6 to 8 hours until the conjunctival hemorrhage resolves.

Chemical injuries

Toxic, acid, and alkaline chemical injuries to the eye can sometimes occur. The severity of the injury caused by ocular burns depends on the concentration, type, and pH of the chemical and on the duration of exposure. Weak acids do not penetrate biologic tissue very well. The hydrogen ion precipitates the protein upon contact and therefore provides some protection to the corneal stroma and intraocular contents. Precipitation of corneal proteins produces a ground-glass appearance in the cornea.

Alkaline solutions and very strong acids penetrate tissues rapidly, causing saponification of the plasma membrane, denaturation of collagen, and vascular thrombosis within the conjunctiva, episclera, and anterior uvea.

Severe pain, blepharospasm, and photophobia are produced by exposure of free nerve endings in the corneal epithelium and conjunctiva. Severe alkaline burns cause an increase in intraocular pressure. Intraocular prostaglandins are released, and the intraocular aqueous pH increases, producing changes in the blood–aqueous barrier and secondary uveitis. Uveitis with anterior synechia formation, eventual chronic glaucoma, phthisis, secondary cataract, and corneal perforation can occur.

Healing of the corneal epithelium is usually accomplished by neovascularization and sliding and increased mitosis of the corneal epithelium. Severe stromal burns within the cornea heal by degradation and removal of necrotic debris, followed by replacement of the collagen matrix and corneal epithelial cells. The release of collagenase, endopeptidase, and cathepsins from polymorphonuclear cells serves to cause further corneal breakdown. In severe cases, only PMNs may be present, and fibroblasts may never invade the corneal stroma.

All chemical burns should be washed copiously with any clean aqueous solution available. If any sticky paste or powder is adherent to the conjunctival sac, remove it with moist cotton swabs and irrigation. Begin mydriasis and cycloplegia by topical application of 1% atropine ophthalmic drops or ointment. Start antibiotic therapy with triple antibiotic ophthalmic ointment or Gentocin ointment every 6 to 8 hours. Treat secondary glaucomas with topical carbonic anhydrase inhibitors. To avoid fibrinous adhesions and symblepharon formation, keep the conjunctival cul-de-sacs free of proteinaceous exudate that can form adhesions. Analgesics are required for pain. Oral nonsteroidal antiinflammatory agents such as carprofen, ketoprofen, meloxicam, or aspirin are recommended.

Persistent epithelial erosions may require a conjunctival flap left in place for 3 to 4 weeks or placement of a topical collagen shield (contact lens). Topical antibiotics, mydriatics, and lubricants (Lacrilube or Puralube ointment) should also be used.

Strong acid or alkali burns can result in severe corneal stromal loss. In the past, topical *N*-acetylcysteine (10% Mucomyst) has been recommended. This treatment is very painful. Other treatments are also available, such as ethylenediaminetetraacetic acid (EDTA) (0.2 M solution) and patient serum to inhibit mammalian collagenase activity. To prepare patient serum, obtain 10 to 12 mL of whole blood from the patient. Spin it down in a serum separator tube after a clot forms and then place the serum in a red-topped tube on the patient's cage. (The contents of the tube are viable for 4 days without refrigeration.) Apply the serum topically to the affected eye every 1 to 2 hours. Avoid using topical steroids because they inhibit fibroblast formation and corneal healing. In severe cases, if conjunctival swelling and chemosis also are present, antiinflammatory doses of oral steroids can be administered short-term. Oral steroids and nonsteroidal antiinflammatory drugs should never be administered to the patient concurrently, because of the risk of gastrointestinal ulcer and perforation.

Corneal abrasions

Corneal abrasions are associated with severe pain, blepharospasm, lacrimation, and photophobia. Animals with such intense pain are often difficult to examine until analgesia has been administered. Topical use of proparacaine (0.5% proparacaine hydrochloride) is usually sufficient to permit relaxation of the eyelids so that the eye can be examined. Using a focal source of illumination and an eye loupe, examine the cornea, inferior and superior conjunctival fornices, and medial aspect of the nictitans for foreign bodies. Place a sterile drop of saline on a fluorescein-impregnated strip and touch the superior conjunctiva once to allow the stain to spread onto the surface of the eye. Irrigate the eye to remove excess stain and then examine the corneal surface for any areas of stain uptake. If an area of the cornea persistently remains green, there is damage to the corneal epithelium in that area.

Initial treatment consists of application of a topical mydriatic (1 drop of 1% atropine in affected eye q12h) to prevent anterior synechiae and improve cycloplegia. Triple antibiotic ointment is the treatment of choice (a ¼-inch strip in the affected eye q8h) until the ulcer heals. In some cases, nonhealing ulcers (e.g., Boxer ulcer, indolent ulcer) form in which the epithelial growth does not adhere to the underlying cornea. Gently debride the loose edges

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of the ulcer/erosion with a cotton swab and topical anesthesia. More severe cases in which only minimal healing has occurred after 7 days of treatment require grid keratectomy, in which a 25-gauge needle is used to gently scratch the surface of the abrasion or ulcer in the form of a grid to promote neovascularization. Apply a topical anesthetic before performing the procedure. A collagen contact lens also may be required to promote wound healing. All corneal abrasions should be reevaluated in 48 hours, and then every 4 to 7 days thereafter until they have healed.

Acute infectious keratitis

Acute infectious keratitis secondary to bacterial infection is characterized by mucopurulent ocular discharge, rapidly progressing epithelial and corneal stromal loss, inflammatory cellular infiltrates into the corneal stroma, and secondary uveitis, often with hypopyon formation. Confirmation of infectious keratitis is based on corneal scrapings and a positive Gram stain. Initial treatment for bacterial keratitis consists of systemic antibiotics and topical ciprofloxacin (0.3% eyedrops or ointment).

Penetrating corneal injury

Penetrating injuries through the cornea may result in prolapse of intraocular contents. Frequently, pieces of uveal tissue or fibrin effectively but temporarily seal the defect and permit the anterior chamber to re-form. Avoid manipulation of these wounds until the animal has been anesthetized, as struggling or excitement can promote loss or dislodgement of the temporary seal and cause the intraocular contents to be extruded.

Superficial corneal lacerations need not be sutured and can be treated the same as a superficial corneal ulcer or abrasion. If the laceration penetrates more than 50% the thickness of the cornea, or extends more than 3 to 4 mm, it should be sutured. When placing sutures in the cornea, it is helpful to use magnification. Referral to a veterinary ophthalmologist is advised. If a veterinary ophthalmologist is not available, use 7-0 or 8-0 silk, collagen, or nylon sutures on a micropoint spatula-type needle. Use a simple interrupted suture pattern and leave the sutures in place for a minimum of 3 weeks. Because many corneal lacerations are jagged and corneal edema forms, most of the wound edges cannot be tightly juxtaposed. In such cases, pull a conjunctival flap across the wound to prevent leakage of aqueous fluid. Never suture through the full thickness of the cornea; rather, the suture should pass through the mid-third of the cornea.

Following closure of the corneal wound, the anterior chamber must be re-formed to prevent anterior synechia formation with secondary glaucoma. Taking care to avoid iris injury, use a 25- or 26-gauge needle to insert sterile saline at the limbus. Any defect in the suture line will be apparent because of leakage of the fluid from the site and should be repaired.

Incarceration of uveal tissue in corneal wounds is a difficult surgical problem. Persistent incarceration of uveal tissue can result in development of a chronic wick in the cornea, a shallow anterior chamber, chronic irritation, edema, vascularization of the cornea, and intraocular infection that can lead to panophthalmitis. Referral to a veterinary ophthalmologist is strongly recommended.

Ocular foreign body

The most common foreign bodies associated with ocular injuries in small animals are bird-shot, BB pellets, and glass. The site of intraocular penetration of the foreign bodies may be obscured by the eyelids. A foreign body entering the eye may penetrate the cornea and fall into the anterior chamber or become lodged in the iris. Foreign bodies may occasionally penetrate the lens capsule, producing cataracts. Some metallic high-speed foreign bodies may penetrate the cornea, iris, and lens to lodge in the posterior wall of the eye or vitreous chamber.

Direct visualization of a foreign body is the best means of localization. Examination of the eye with an indirect ophthalmoscope or biomicroscope (if available) is invaluable for locating foreign bodies. Indirect visualization of the ocular foreign body can also be achieved through radiographic techniques. Three separate views should be obtained to

determine the plane of location of the foreign object. CT or MRI may prove useful, although scatter from the foreign body may make it difficult to directly visualize with these techniques. Ocular ultrasound is perhaps the most useful and refined radiographic technique for locating intraocular foreign bodies.

Before removing any foreign body from the eye, the risk and surgical danger of removing it must be weighed against the risks of leaving it in place. Metallic foreign bodies in the anterior chamber are much easier to remove than nonmagnetic ones. Attempted removal of foreign objects from the vitreous chamber of the eye has consistently produced poor results. For the best chance of recovery, ocular foreign bodies should be removed by a veterinary ophthalmologist whenever possible.

Ocular trauma

Blunt trauma to the globe can result in luxation or subluxation of the lens. The subluxated lens may move anteriorly and make the anterior chamber more shallow. Trembling of the iris (iridodonesis) may be noticed when the lens is subluxated. In complete luxation, the lens may fall totally into the anterior chamber and obstruct aqueous outflow, causing secondary glaucoma. Alternatively, the lens may be lost into the vitreous cavity. Luxation of the lens is almost always associated with rupture of the hyaloid membrane and herniation of the vitreous through the pupillary space.

Emergency surgery for lens luxation is required if the lens is entirely within the anterior chamber or incarcerated within the pupil, causing a secondary pupillary block glaucoma. Acute elevation in intraocular pressure can cause vision loss within 48 hours; thus, lens removal should be accomplished as quickly as possible. Referral to a veterinary ophthalmologist is recommended.

Severe trauma to the globe or a direct blow to the head can result in retinal or vitreous hemorrhage. There may be large areas of subretinal or intraretinal hemorrhage. Subretinal hemorrhage assumes a discrete globular form, and the blood appears reddish-blue in color. The retina is detached at the site of hemorrhage. Superficial retinal hemorrhage may assume a flame-shaped appearance, and preretinal or vitreous hemorrhage assumes a bright-red amorphous appearance, obliterating the underlying retinal architecture. Retinal and vitreous hemorrhage secondary to trauma usually resorbs spontaneously over a 2- to 3-week period. Unfortunately, vitreous hemorrhage, as it organizes, can produce vitreous traction bands that eventually produce retinal detachment.

Expulsive choroid hemorrhage can occur at the time of injury and usually leads to retinal detachment, severe visual impairment, and total loss of vision. Treatment of vitreal and retinal hemorrhage includes rest and correction of factors that may predispose to intraocular hemorrhage. More complicated cases may require vitrectomy performed by a veterinary ophthalmologist.

Hyphema

Hyphema refers to blood in the anterior chamber of the eye. The most common traumatic cause of hyphema is an automobile accident. Hyphema may also present because of penetrating ocular wounds and coagulopathies. Blood within the eye may come from the anterior or posterior uveal tract. Trauma to the eye may result in iridodialysis or a tearing of the iris at its root, permitting excessive bleeding from the iris and ciliary body. Usually, simple hyphema resolves spontaneously in 7 to 10 days and does not cause vision loss. Loss of vision following bleeding into the anterior chamber is associated with secondary ocular injuries such as glaucoma, traumatic iritis, cataract, retinal detachment, endophthalmitis, and corneal scarring.

Treatment of hyphema must be individualized, but there are severe general principles of treatment. First, stop ongoing hemorrhage and prevent further bleeding whenever possible. This may involve correction of the underlying cause, if a coagulopathy is present. Next, aid in the elimination of blood from the anterior chamber, control secondary glaucoma, and treat associated injuries, including traumatic iritis. Finally, detect and treat any late complications of glaucoma.

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In most cases of traumatic hyphema, little can be done to arrest or prevent ongoing hemorrhage. It is best to restrict the animal's activity and prohibit exertion. Rebleeding can occur within 5 days, and intraocular pressure must be monitored closely. After 5 to 7 days, the blood in the anterior chamber will change color from a bright red to bluish-black ("eight-ball hemorrhage"). If total hyphema persists and intraocular pressure rises despite therapy, surgical intervention by a veterinary ophthalmologist may be necessary.

The primary route of escape of RBCs from the anterior chamber is via the anterior drainage angle. Iris absorption and phagocytosis play a minor role in the removal of blood from the anterior chamber. Because of the associated traumatic iritis in hyphema, topical administration of a glucocorticoid (1% dexamethasone drops or 1% prednisolone drops) is advised to control anterior chamber inflammation. A cycloplegic agent (1% atropine) should also be used.

The formation of fibrin in the anterior chamber of the eye secondary to hemorrhage can produce adhesions of the iris and secondary glaucoma (see section on Glaucoma Secondary to Hyphema) by blocking the trabecular network. Hyphema secondary to retinal detachment (Collie ectasia syndrome) and end-stage glaucoma are extremely difficult to treat medically and have a poor prognosis.

Proptosis

Proptosis of the globe is common secondary to trauma, particularly in brachycephalic breeds. Proptosis of the globe in dolichocephalic breeds requires a greater degree of initiating contusion than the brachycephalic breeds because the orbits are so much deeper. Therefore, secondary damage to the eye and CNS associated with proptosis of the globe may be greater in the Collie or Greyhound than in the Pug.

When proptosis occurs, carefully evaluate the cardiovascular system for evidence of hypovolemic or hemorrhagic shock. Examine the respiratory and neurologic systems. Be sure to establish an airway and treat shock, if present. Control hemorrhage and stabilize the cardiovascular system before attempting to replace the globe within its orbit or perform enucleation. During the initial management of the cardiovascular and respiratory systems, the eye should be covered with an ophthalmic grade ointment or sponges soaked in sterile saline to prevent the globe from drying out. Proptosis of the globe can be associated with serious intraocular problems including iritis, chorioretinitis, retinal detachment, lens luxation, and avulsion of the optic nerve.

Stain the surface of the eye with fluorescein to look for topical abrasions or ulcers. Carefully examine the sclera, cornea, and conjunctiva for penetrating injuries that may allow aqueous leakage. Evaluate the size, location, and response to light of the pupil. A reactive pupil is better than a mydriatic fixed pupil. Topical administration of a mydriatic (atropine 1%) to prevent persistent miosis and synechia formation is indicated, along with topical and oral antibiotics and oral analgesic therapy.

Reposition the proptosed globe with the patient under general anesthesia. Make a lateral canthotomy incision to widen the palpebral fissure. Lavage the globe with sterile saline irrigation to remove any external debris. Place a copious amount of triple antibiotic ophthalmic ointment on the surface of the eye and then gently press the globe into the orbit using the flat side of a scalpel handle or a moistened sterile surgical sponge. Do not probe the retro-orbital space with a needle or attempt to reduce intraocular pressure by paracentesis. When the globe is replaced in the orbit, close the lateral canthotomy incision with simple interrupted sutures. Place three non-penetrating mattress sutures in the lid margins but do not draw them together. Tighten the lid sutures through small pieces of a red rubber catheter or length of intravenous extension tubing to prevent the sutures from causing lid necrosis. Leave the medial canthus of the eye open in order to allow topical treatment.

Postoperative treatment is directed at preventing further iritis and preventing infection. Administer systemic broad-spectrum antibiotics (Clavamox, 16.25 mg/kg PO bid) and analgesic drugs. Apply topical triple antibiotic ophthalmic ointment (1/4 inch in affected eye q6-8h) and atropine (1% in affected eye q12h) to prevent infection, cycloplegia, and anterior synechiae. Antiinflammatory doses of systemic steroids can also be added to the treatment

if severe periorbital inflammation is present. Systemic steroids should never be used in conjunction with nonsteroidal antiinflammatory drugs, because of the risk of gastrointestinal ulceration and perforation.

The sutures should remain in place for a minimum of 3 weeks. After this time, remove the sutures and inspect the globe. If proptosis recurs, repeat the treatment.

Following proptosis, strabismus is common secondary to periorbital muscle injury. Even after extensive treatment, vision in the eye may still be lost. Nonvisual eyes can remain in place, but phthisis may develop.

Glaucoma secondary to hyphema

Carbonic anhydrase inhibitors such as acetazolamide and dichlorphenamide decrease aqueous secretion and may effectively reduce intraocular pressure if the trabecular outflow is still functioning at 40% of its capacity. An eye with a poorly functional trabecular outflow system will respond poorly to therapy with carbonic anhydrase inhibitors. Osmotic agents such as mannitol or glycerol may be helpful in controlling glaucoma secondary to hyphema. Reduction in vitreous chamber size can make the anterior chamber deeper and may allow increased aqueous outflow. Evacuation of blood or blood clots from the anterior chamber is not advisable unless the glaucoma cannot be controlled medically or there is no indication after a prolonged period of time that blood is being resorbed.

Tissue plasminogen activator (t-PA) has proved to be useful in may be helpful in lysing blood clots and preventing excessive fibrin formation. The t-PA is reconstituted to make a solution of 250 μ /mL, which is then frozen at -70° C in 0.5-mL aliquots. The thawed, warmed reconstituted t-PA is injected into the anterior chamber.

Blind probing of the anterior chamber of the eye and surgical intervention in an attempt to remove blood clots can cause serious complications such as rebleeding, lens luxation, iris damage, and damage to the corneal epithelium, and therefore is not advised.

Acute glaucoma

Acute glaucoma is a rise in intraocular pressure that is not compatible with normal vision. Glaucoma may present as early acute congestive or noncongestive glaucoma, or as end-stage disease. Cardinal signs of glaucoma are a sudden onset of pain, photophobia, lacrimation, deep episcleral vascular engorgement, edematous insensitive cornea, shallow anterior chamber depth, dilated unresponsive pupil, loss of visual acuity, and buphthalmia. Intraocular pressure usually exceeds 40 mm Hg but may be normal or only slightly increased if glaucoma is secondary to anterior uveitis.

Most forms of clinical glaucoma in dogs are secondary to some other intraocular problem. Primary glaucoma is recognized in some breeds, including the Bassett Hound, Cocker Spaniel, Samoyed, Bouvier des Flandres, and some Terrier breeds either from goniodysgenesis or a predisposition to lens luxation. Other common causes of acute glaucoma are anterior uveitis and intumescent lens secondary to rapid cataract development, particularly in dogs with diabetes mellitus.

Treatment involves investigation of the underlying cause of the sudden rise in intraocular pressure and rapid reduction in intraocular pressure. Permanent visual impairment is often associated with chronically buphthalmic globes or the presence of rippling or striae formation on the cornea. Referral to a veterinary ophthalmologist is recommended.

If the eye is still visual and not buphthalmic, the prognosis is favorable, depending on the cause of the acute glaucoma. Treatment to reduce intraocular pressure consists of improving aqueous outflow, reducing intraocular volume with osmotic agents, and reducing aqueous formation (Table 1-47).

The use of topical mydriatic agents in acute glaucoma is contraindicated because of the risk of making lens luxation or anterior uveitis worse. Referral to a veterinary ophthalmologist for emergency surgery is indicated in cases of iris bombe, intumescent lens, or lens subluxation.

Administer osmotic agents to reduce the size of the vitreous body and the amount of aqueous. Osmotic agents create an osmotic gradient between the intraocular fluids and the

TABLE 1 - 47 Drugs Indicated for the Emergency Management of Acute Glaucoma

Osmotic agents

Mannitol	1-2 g/kg IV over 20-60 minutes
Glycerol	1.4 g/kg PO; watch for vomiting

Carbonic anhydrase inhibitors

Dichlorphenamide (Daranide)*	2-10 mg/kg PO q12-24h
Methazolamide (Neptazene)*	5-10 mg/kg PO q8-12h
Dorzolamide (Trusopt)*	1 drop topical q8-12h

β-Blocker

Timolol maleate (Timoptic 0.25 or 0.5%)*	1 drop topical q12h
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Prostaglandin analogue

Latanaprost‡	1 drop topical q24h
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*Vomiting, diarrhea, panting, staggering, and disorientation are side effects.

†Do not use in cats with bronchitis (asthma).

‡Do not use if uveitis or lens subluxation is present.

vascular bed, thus allowing osmotic removal of fluid independent of the aqueous inflow and outflow systems. If no other treatments are available, oral glycerol (50%, 0.6 mL/kg or 1.4 g/kg) can be used to effectively reduce intraocular pressure. An adverse side effect of oral glycerol treatment is protracted vomiting. Do not use glycerol in a diabetic patient. Mannitol (1-2 g/kg IV over 1 hour) also effectively reduces intraocular pressure but does not cause vomiting.

Carbonic anhydrase inhibitors can be used to reduce intraocular volume by reducing aqueous production. Oral administration of dichlorphenamide, methazolamide, and acetazolamide (2-4 mg/kg) is usually not very effective alone in reducing aqueous volume and intraocular pressure and also can cause metabolic acidosis. Topical carbonic anhydrase inhibitors appear to be more effective (dorzolamide, Trusopt) when used in conjunction with topical beta-blockers (timolol, 0.25% or 0.5% solution q8h). The most effective treatment for acute pressure reduction is use of a topical prostaglandin inhibitor (latanaprost). Usually just one or two drops effectively reduces intraocular pressure in the emergency stages, until the patient can be referred to a veterinary ophthalmologist the following day.

Additional Reading

- Abrams KL: Medical and surgical management of the glaucoma patient. Clin Tech Small Anim Pract 16:71-76, 2001.
- Blocker T, van der Woerd A: The feline glaucomas: 82 cases (1995-1999). Vet Ophthalmol 4: 81-85, 2001.
- Gellatt KN, Brooks DE: The canine glaucomas. In Gellatt KN (ed): Veterinary ophthalmology. 3rd Edition. Lippincott, Baltimore, 1999.
- Gilger BC, Hamilton HL, Wilkie DA, et al: Traumatic ocular protrusion in dogs and cats: 84 cases. J Am Vet Med Assoc 206(8):1186-1189, 1995.
- Gionfriddo JR, Powell CC: Traumatic glaucoma in a dog. Vet Med 96(11):830-836, 2001.
- Grahn BH, Szentimrey D, Pharr JW, et al: Ocular and orbital porcupine quills in the dog: a review and case series. Can Vet J 36(8):488-493, 1995.
- Komaromy AM, Ramsey DT, Brooks DE, et al: Hyphema: pathophysiologic considerations. Comp Cont Educ Pract Vet 21(11):1064-1069, 1999.
- Mandell D: Ophthalmic emergencies. Clin Tech Small Anim Pract 15(2):94-100, 2000.
- Singh A, Cullen CL, Grahn BH: Alkali burns to the right eye. Can Vet J 45(9):777-778, 2004.
- van der Woerd A: The treatment of acute glaucoma in dogs and cats. J Vet Emerg Crit Care 11(3):199-205, 2001.

ONCOLOGIC EMERGENCIES

Many clinical conditions that are presented as emergencies may be due in part or wholly to the presence of a neoplasm. Paraneoplastic signs are summarized in Table 1-48. Prompt identification of the neoplasia combined with knowledge of treatment, expected response to therapy, and long-term prognosis can aid owners and practitioners in making appropriate treatment decisions.

HEMORRHAGE OR EFFUSION

Hemorrhage or effusion can occur in any body cavity as a result of the presence of benign or malignant tumors. Tumors secrete anticoagulants to allow angiogenesis to grow unchecked. Hemorrhage often occurs as a result of rupture of a neoplasm or invasion of a neoplasm into a major vascular structure. Effusion may be the result of direct fluid production by the mass or may be due to obstruction of lymphatic or venous flow.

TABLE 1 - 48 Paraneoplastic Syndromes in Dogs and Cats

Paraneoplastic syndrome	Cause/Clinical signs	Tumor type	Treatment
Neutropenia	Immunosuppression, chemotherapy, leukemia and myelophthisis, fever, hypothermia	Lymphoma (stage V), leukemia, multiple myeloma	Granulocyte colony-stimulating factor (G-CSF), antibiotics
Sepsis	Cellular immune dysfunction, indwelling intravenous and urinary catheters, weakness, collapse, fever, vomiting, diarrhea, hypotension, lethargy, melena	Various	G-CSF, intravenous fluids, antibiotics
Thrombocytopenia	Decreased bone marrow production (chemotherapy, hyperestrogenism), increased destruction with microangiopathic disease, disseminated intravascular coagulation (DIC), blood loss from tumor, immune-mediated destruction, petechiae and ecchymosis	Lymphoma, multiple myeloma, hemangiosarcoma, leukemia, gastrointestinal adenocarcinoma, any tumor type	Blood transfusion, treatment of underlying disease
Anemia	Decreased bone marrow production, hemorrhage, microangiopathic disease, DIC, immune-mediated destruction, chemotherapy, weakness, lethargy, tachycardia, tachypnea	Leukemia, lymphoma, hyperestrogenemia, adenocarcinoma, thyroid carcinoma	Blood transfusion, treatment of underlying disease

Continued

TABLE 1 - 48 Paraneoplastic Syndromes in Dogs and Cats—cont'd

Paraneoplastic syndrome	Cause/Clinical signs	Tumor type	Treatment
Erythrocytosis	Erythropoietin production by tumor or renal hypoxia, lethargy, dementia, vomiting, renal azotemia	Renal carcinoma, lymphoma, primary polycythemia vera	Identification and treatment of underlying cause, hydroxyurea, phlebotomy
DIC	Microangiopathic syndrome	Many	Blood transfusion, heparin, fresh frozen plasma
Hypergammaglobulinemia	Increased serum viscosity following increased immunoglobulin G production by tumor, ocular hemorrhage and retinal detachment, dementia, seizures, petechiae, bleeding, occult infection	Plasma cell tumor, multiple myeloma	Treatment of underlying cause, melphalan and prednisone
Acute tumor lysis syndrome	Acute tumor cell death after chemotherapy, acute collapse and shock, vomiting, atrial standstill from hyperkalemia, bradycardia, muscle twitching	Lymphoma, leukemia	Crystalloid fluid therapy, treatment of hyperkalemia, monitoring of electrolyte status
Hypercalcemia	Parathyroid-related peptide increased osteoclast activity; vomiting, diarrhea, constipation, polyuria/polydipsia, bradycardia, stupor, hypertension, weakness, seizures	Lymphoma, apocrine gland adenocarcinoma, multiple myeloma, mammary adenocarcinoma, parathyroid adenoma, parathyroid adenocarcinoma	Administration of 0.9% sodium chloride intravenously, prednisolone, bisphosphonates, furosemide, salmon calcitonin
Hypoglycemia	Sepsis, insulin secretion or insulin-like peptide secretion from tumor, catecholamine release, weakness, seizures	Pancreatic beta cell tumor (insulinoma), leiomyosarcoma, leiomyoma, oral melanoma, hepatoma, hepatocellular carcinoma	Surgical removal of tumor, supplemental dextrose in intravenous fluids, parathyroid hormone-related peptide, prednisone, diazoxide, propranolol

Hemorrhagic effusions in the abdominal cavity occur most commonly with neoplastic masses of the spleen or liver. The most common causes are hemangiosarcoma and hepatocellular carcinoma. Clinical signs associated with acute abdominal hemorrhage, regardless of the cause, are related to hypovolemic shock and decreased perfusion and include pale mucous membranes, tachycardia, anemia, lethargy, and acute collapse. Treatment for abdominal hemorrhage includes placement of a large-bore peripheral cephalic catheter and starting one fourth of a shock dose (90 mL/kg/hour for dogs, and 44 mL/kg/hour for cats) of intravenous crystalloid fluids, taking care to carefully monitor perfusion parameters of heart rate, capillary refill time, mucous membrane color, and blood pressure. Administer intravenous colloids such as Dextran-70, Hetastarch, and oxyglobin (5-10 mL/kg IV bolus) to restore intravascular volume and normotension. Treat severe anemia with whole blood or packed RBCs to improve oxygen-carrying capacity and oxygen delivery (see sections on Transfusion Medicine and Treatment of Shock). Confirm the presence of hemoabdomen abdominocentesis (see section on Abdominocentesis). The presence of nonclotting hemorrhagic effusion is consistent with free blood. Packed cell volume of the fluid is usually the same or higher than that of the peripheral blood. An abdominal compression bandage can be placed while further diagnostics are being performed.

In cases of acute hemoabdomen, obtain right lateral, left lateral, and ventrodorsal or dorsoventral thoracic radiographs to help rule out obvious metastasis. Monitor the patient's ECG and correct dysrhythmias as necessary (see section on Cardiac Dysrhythmias). Surgery is indicated once the patient is stabilized. In some cases, hemorrhage is so severe that the patient should be taken immediately to surgery.

When recommending surgery for a hemorrhaging intraabdominal mass, it is important to discuss likely diagnoses and long-term prognosis with the owner. Hemangiosarcoma usually involves the spleen or liver or both. The presence of free abdominal hemorrhage is associated with a malignant tumor in 80% of cases. Even when free abdominal hemorrhage is not present, the tumor is malignant in 50% of cases. Approximately 66% (two thirds) of masses in the spleen are malignant (hemangiosarcoma, lymphoma, mast cell tumor, malignant fibrous histiocytoma, leiomyosarcoma, fibrosarcoma), and approximately one third are benign (hematoma, hemangioma).

Hepatocellular carcinoma usually affects one liver lobe (usually the left), and surgery is the treatment of choice. With complete surgical excision, median survival in dogs is longer than 300 days. If diffuse disease is observed at the time of surgery, the prognosis is poor.

Nonhemorrhagic effusions are associated with mesothelioma, lymphoma, carcinomatosis, or any mass that causes vascular or lymphatic obstruction. Clinical signs of respiratory distress and abdominal distention with nonhemorrhagic effusions are usually slowly progressive in onset and not as severe as those observed with hemorrhage. Treatment is usually aimed at identification of the underlying cause.

Obtain a fluid sample via thoracocentesis or abdominocentesis. To obtain further cells for cytologic evaluation, aspirate fluid from the thoracic or abdominal mass with ultrasound guidance. Cytologic evaluation of the fluid will often elucidate the causative tumor type. An abdominal ultrasound can determine the degree of metastasis. Perform therapeutic abdominocentesis or thoracocentesis if the effusion is causing respiratory difficulty. Rapid re-accumulation of the fluid potentially can cause hypoproteinemia and hypovolemic shock.

Mesothelioma is a rare tumor most commonly observed in urban environments. In humans, mesothelioma has been associated with exposure to asbestos. It is sometimes difficult to differentiate between reactive mesothelial cells and malignant mesothelial cells. Treatment is aimed at controlling the neoplastic effusion. Intracavitary cisplatin has been demonstrated to slow rates of fluid re-accumulation, but is largely a palliative therapy. Lymphoma is another tumor type that can cause thoracic or abdominal effusion. Cytologic evaluation of the fluid usually reveals abundant lymphoblasts. Treatment with multiagent chemotherapy protocols, with or without adjunctive radiation therapy, can prevent tumor remission and stop fluid accumulation.

Carcinomatosis occurs as a result of diffuse seeding of the abdominal cavity with malignant carcinomas and has a poor prognosis. Carcinomatosis may occur de novo or from

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metastasis of a primary tumor. Treatment consists of fluid removal when respiratory difficulty occurs, with or without intracavitary cisplatin as a palliative measure. Cisplatin should never be used in cats due to fatal acute pulmonary edema.

THORACIC CAVITY

Clinical signs of hemorrhagic thoracic effusion include acute respiratory distress, anemia, hypovolemic or cardiogenic shock, and collapse. Hemorrhagic thoracic effusions are rare in association with neoplastic effusions. A notable exception is intrathoracic hemorrhage in young dogs with osteosarcoma of the rib. Hemorrhage can result when a primary lung tumor erodes through a vessel. Hemangiosarcoma of the lungs or right auricular area can also result in hemorrhagic thoracic effusion. In many cases, hemorrhage may be confined to the pericardial sac with a right auricular mass, causing a globoid cardiac silhouette on thoracic radiographs.

Treatment consists of pericardiocentesis (see section on Pericardial Effusion and Pericardiocentesis) and placement of a pericardial window, or the mass may be removed if it is in the right auricular appendage and resectable. Although surgery can resolve clinical signs of right-sided heart failure, metastatic disease often develops soon afterward.

Nonhemorrhagic thoracic effusion is more common than hemorrhagic thoracic effusion, and is caused most commonly by mesothelioma, lymphoma, carcinomatosis, and thymoma. Clinical signs develop gradually and include respiratory difficulty, cyanosis, and cough. Supplemental oxygen should be administered. In many cases, thoracocentesis can be therapeutic and diagnostic. Obtain thoracic radiographs both before and after thoracocentesis to determine whether a mass effect is present. Following identification of a cause, definitive therapy can be instituted.

Mesotheliomas are rare and are associated with diffuse serosal disease. They are more common in dogs than in cats. Effusions caused by mesotheliomas can affect the pleural or pericardial cavities. Treatment is directed at removing effusion fluid and controlling reaccumulation with use of intracavitary platinum compounds, carboplatin, and cisplatin can be used in dogs. (Cisplatin and carboplatin should never be used in cats.) Chemical or physical pleurodesis may be helpful in controlling reaccumulation of fluid, but it is very painful in small animal patients.

Thoracic effusion secondary to lymphoma often is associated with an anterior mediastinal mass. T-cell lymphoma is the most common type of mediastinal mass observed in dogs. B-cell lymphoma is associated with a decreased response to chemotherapy and shorter survival times. Treatment consists of combination chemotherapy with or without radiation therapy to decrease mass size.

Carcinomatosis is a diffuse disease of the pleural cavity that often is a result of metastasis from a primary pulmonary carcinoma or mammary adenocarcinoma. Treatment is similar to that for mesothelioma and is aimed at controlling the effusion and delaying its recurrence.

Thymomas have been documented in both dogs and cats. Dogs most commonly present with a cough, while cats present with clinical signs of respiratory distress and a restrictive respiratory pattern associated with the presence of pleural effusion. An anterior mediastinal mass is often observed on thoracic radiographs. In some cases, the pleural effusion must be drained via thoracocentesis before a mass is visible. Ultrasound-guided aspiration and cytologic evaluation of the mass reveal a malignant epithelial tumor with small lymphocytes and mast cells. Prognosis is good if the tumor can be completely excised. Treatment consists of surgical removal with or without presurgical radiation therapy to shrink the mass. Paraneoplastic syndromes of myasthenia gravis have been documented in dogs with thymomas. If megaesophagus or aspiration pneumonia is present, the prognosis is more guarded because of the high rate of complications.

NEOPLASIA CAUSING ORGAN SYSTEM OBSTRUCTION**Urinary tract**

Obstructive lesions affecting the urinary tract can be extramural (intra-abdominal, pelvic, or retroperitoneal) or intramural (urethral, bladder, or urethral wall). Transitional cell

carcinoma is the most common type of bladder tumor observed in dogs. Prostatic adenocarcinoma, or neoplasia of the sublumbar lymph nodes (lymphoma, adenocarcinoma from apocrine gland adenocarcinoma), also can cause urethral obstruction. Treatment is aimed at relieving the obstruction and then attempting to identify the cause of the disease. To alleviate the obstruction, pass a urinary catheter whenever possible. Perform cystocentesis only as a last resort because of the risk of seeding the peritoneal cavity with tumor cells if transitional cell carcinoma is the cause of the obstruction. Institute supportive therapy including intravenous fluids and correction of electrolyte abnormalities.

Plain radiographs may reveal a mass lesion or may not be helpful without double contrast cystography. Abdominal ultrasound is more sensitive in identifying a mass lesion in the urinary bladder. Masses in the pelvic urethra are difficult to visualize with ultrasonography. Double contrast cystourethrography is preferred. Once the patient is stabilized, biopsy or surgery is indicated to identify the cause of the mass and attempt resection. Urine tests for transitional cell carcinoma are available for identification of transitional cell carcinoma in the dog.

Complete surgical excision of transitional cell carcinoma or removal of benign tumors of the urinary bladder yields a favorable prognosis. Poorer prognosis is seen with incomplete excision. Many transitional cell carcinomas are located in the trigone region of the bladder and cannot be completely excised. The nonsteroidal antiinflammatory drug piroxicam is helpful in alleviating clinical signs for a reported 7-month median survival. In some dogs, cisplatin and carboplatin may delay recurrence of transitional cell carcinoma.

Tumors of the prostate gland are always malignant and occur with equal frequency in castrated and uncastrated male dogs. Diagnosis of prostatic tumors is based on ultrasonographic evidence of a mass effect or prostatomegaly and on transrectal or transabdominal aspiration or biopsy. Surgery, chemotherapy, and radiation therapy generally are unrewarding over the long term, although palliative radiation therapy may relieve clinical signs for 2 to 6 months.

Gastrointestinal obstruction

Luminal tumors of the gastrointestinal tract typically cause obstruction, with slowly progressive clinical signs including vomiting, inappetence, and weight loss, or with acute severe protracted vomiting. Extraluminal obstructive lesions usually arise from adhesions, or strangulation may occur, resulting in obstruction. Perforation of the mass through the gastric or intestinal wall can cause peritonitis. Treatment consists of initial stabilization and rehydration, evaluation for evidence of metastasis, and surgical resection of the affected area in cases of adenocarcinoma, leiomyoma, leiomyosarcoma, and obstructive or perforated lymphoma.

Gastric and intestinal adenocarcinoma are the most common gastrointestinal tumors observed in dogs. Affected animals typically have a history of anorexia, weight loss, and vomiting. Obtain an abdominal ultrasound before performing any surgery. Fine needle aspirates of the mass and adjacent lymph nodes are usually diagnostic and can determine whether there is local metastasis. Many tumors are not resectable, and metastasis occurs in approximately 70% of cases. Dogs with smaller tumors that can be resected typically have longer survival times.

Leiomyosarcomas occur in the intestines of dogs, and carry a more favorable prognosis than adenocarcinoma if the mass can be completely resected. With complete resection, the average survival time is longer than 1 year. The paraneoplastic syndrome of hypoglycemia has been observed with this tumor type.

Gastrointestinal lymphoma is the most common tumor of the gastrointestinal tract observed in cats. In comparison, it is relatively rare in dogs. Unless there is complete obstruction or perforation of the gastrointestinal tract, surgical treatment for gastrointestinal lymphoma is not indicated. Rather, multiple chemotherapy drugs are used in combination to achieve remission and resolution of the clinical signs of anorexia, weight loss, and vomiting. Treatment responses unfortunately are poor.

Mast cell tumors of the gastrointestinal tract typically are manifested as gastrointestinal ulceration and hemorrhage in up to 83% of patients. The gastrointestinal hemorrhage that

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occurs with mast cell tumors results from increased acid secretion as a result of histamine receptor stimulation. Treatment consists of histamine or proton pump inhibition (ranitidine, famotidine, cimetidine, or omeprazole). Bowel perforation is a rare complication.

PARANEOPLASTIC SYNDROMES

Chemotherapy-related toxicities

Many chemotherapy agents exert their effects on rapidly dividing normal and neoplastic cells. Normal tissues that are commonly affected include the bone marrow, gastrointestinal tract, skin and hair follicles, and reproductive organs. Some drugs have unique organ-specific toxicities that must be monitored. Knowledge and recognition of the expected type and onset of complications can alleviate their severity by rapid treatment, when complications occur (see Table 1-48).

Bone marrow toxicity

Neutropenia is the most common bone marrow toxicity observed secondary to chemotherapy in small animal patients (Table 1-49). In most cases, the neutropenia is dose-dependent. The nadir, or lowest neutrophil count, is typically observed 5 to 10 days after chemotherapy treatment. Once the nadir occurs, bone marrow recovery is observed, with an increase in circulating neutrophils within 36 to 72 hours (Table 1-49).

Treatment of myelosuppression is largely supportive to treat or prevent sepsis. Prophylactic antibiotics are recommended in the afebrile patient with a neutrophil count <2000/ μ L. Acceptable antibiotics include trimethoprim-sulfa and amoxicillin-clavulanate. Granulocyte-colony stimulating factor (G-CSF) (e.g., Neupogen) is a recombinant human product that stimulates the release of neutrophils from the bone marrow, and its use shortens the recovery time following myelosuppressive drug therapy. Disadvantages of G-CSF include antibody production in response to the drug within 4 weeks of use and its high cost. To prevent ongoing neutropenia, subsequent chemotherapy dosages should be decreased by 25%, and the interval in between treatments increased. Whenever possible, overlap of myelosuppressive drugs should be avoided.

Gastrointestinal toxicity

Acute gastrointestinal toxicity can occur within 6 to 12 hours after administration of cisplatin and actinomycin D. In many cases, pretreatment with the antiemetics metoclopramide, butorphanol, chlorpromazine, dolasetron or ondansetron can prevent chemotherapy-induced nausea and vomiting. Vomiting can also occur as a delayed side effect 3 to 5 days after treatment with doxorubicin (Adriamycin), actinomycin D, methotrexate, and Cytoxan. In delayed reactions, vomiting and diarrhea are caused by damage to intestinal crypt cells. Treatment consists of administration of antiemetics, intravenous fluids, and a bland highly digestible diet. Doxorubicin also can cause hemorrhagic colitis within 5 to 7 days of administration. Treatment includes a bland diet, metronidazole, and tylosin tartrate (Tylan Powder).

TABLE 1 - 49 Classification Scheme for Myelosuppression Associated with Chemotherapy

Degree of myelosuppression	Time of nadir	Causative agent
Mild to none	Not observed	Vincristine (low-dose), L-asparaginase, glucocorticosteroids
Moderate	7-10 days	Melphalan, cisplatin, mitoxantrone, actinomycin D
Severe	7-10 days	Doxorubicin, cyclophosphamide, vinblastine

Paralytic ileus can be observed 2 to 5 days after administration of vincristine. This side effect is more common in humans than animals and can be treated with metoclopramide once a gastrointestinal obstruction has been ruled out.

Cardiotoxicity

Doxorubicin (Adriamycin) causes a dose-dependent dilative cardiomyopathy when the cumulative dose reaches 100 to 150 mg/m². In many cases, however, clinical signs do not occur until the cumulative dose is 240 mg/m². The myocardial lesions are irreversible. Treatment of cardiac dysrhythmias is dependent on the type of dysrhythmia (see section on Treatment of Dysrhythmias). Discontinue doxorubicin and administer diuretics and positive inotropic therapy for dilative cardiomyopathy in order to delay the progression of congestive heart failure (see sections on Treatment of Congestive Heart Failure). If abnormalities are shown on electrocardiography performed before beginning therapy, substitute liposome-encapsulated doxorubicin or mitoxantrone substituted in the chemotherapy protocol. Cardioprotectant drugs such as vitamin E, selenium, and N-acetyl cysteine have shown some promise in the prevention of doxorubicin-induced cardiotoxicity.

Urinary bladder toxicity

Cyclophosphamide can cause a sterile hemorrhagic cystitis. Damage to the urinary bladder mucosa and vessels is caused by the toxic metabolite acrolein. Clinical signs of sterile hemorrhagic cystitis include a history of cyclophosphamide administration, stranguria, hematuria, and pollakiuria. Treatment for sterile hemorrhagic cystitis is discontinuation of the drug, treatment of any underlying urinary tract infection with antibiotic therapy based on susceptibility testing, and intravesicle drug administration. In extremely refractory cases, surgical debridement and cauterization of the bladder mucosa may be necessary.

Prevention of sterile hemorrhagic cystitis includes emptying the bladder frequently and administering the drug in the morning. Concurrent administration of prednisone can induce polyuria and polydipsia. If sterile hemorrhagic cystitis occurs, chlorambucil can be substituted as a chemotherapeutic agent.

Anaphylactic reactions

Anaphylactic reactions have been observed with the administration of L-asparaginase, Adriamycin, etoposide, and paclitaxel. The risk of anaphylaxis increases with repeated administration, although in some animals anaphylaxis will occur on the first exposure to the drug. Treatment consists of administration of epinephrine, diphenhydramine, famotidine, and glucocorticosteroids, as with any other life-threatening allergic reaction (see section on Treatment of Allergic Reactions). To decrease the risk of an adverse reaction, give diphenhydramine (2.2 mg/kg IM) 15 to 30 minutes before drug administration. Slowing the rate of intravenous infusion also can decrease the chance of an anaphylactic reaction.

Species-specific toxicities

Cisplatin can cause a fatal irreversible pulmonary edema in cats, even at low dosages. 5-Fluorouracil (5-FU) can cause a severe neurotoxicity in cats that results in ataxia and seizures. Never use cisplatin or 5-FU in cats.

Additional Reading

- Henry CJ: Management of transitional cell carcinoma. *Vet Clin North Am Small Anim Pract* 33(3):597-613, 2003.
- Henry CJ, Tyler JW, McEntee MC, et al: Evaluation of a bladder tumor antigen test as a screening test for transitional cell carcinoma of the lower urinary tract in dogs. *Am J Vet Res* 64(8):1017-1020, 2003.
- Liptak JM, Brutscher SP, Monnet E, et al. Transurethral resection in the management of urethral and prostatic neoplasia in 6 dogs. *Vet Surg* 33(5):505-516, 2004.

1

Nyland TG, Wallack ST, Wisner ER: Needle tract implantation following US-guided fine-needle aspiration biopsy of transitional cell carcinoma of the bladder, urethra, and prostate. *Vet Radiol Ultrasound* 43(1):50-53, 2002.

Ogilvie GK, Moore AS: *Managing the Veterinary Cancer Patient. A Practice Manual.* Veterinary Learning Systems, Trenton, NJ, 1997.

Rocha TA, Mauldin GN, Patnaik AK, Bergman PJ: Prognostic factors in dogs with urinary bladder carcinoma. *J Vet Intern Med* 14(5):486-490, 2000.

Walters JM, Connally HE, Ogilvie GK, et al: EM. Emergency complications associated with chemotherapeutics and cancer. *Comp Contin Educ Pract Vet* 25(9):676-688, 2003.

POISONS AND TOXINS

Poisoning cases benefit from a rapid, organized approach. Key points in this approach are giving appropriate advice over the telephone, being able to access information sources, and providing appropriate treatment. There are only a few classes of poisons that account for the majority of toxicities reported in dogs and cats.

Every veterinarian should develop a familiarity with the clinical management of rodenticide and insecticide toxicity and be prepared with antidotes on hand. Beyond the most common toxins, the spectrum of possibilities is endless, and the veterinarian must rely on appropriate information resources. It is important to have available a comprehensive source of pharmaceutical and plant identification resources.

Remarkably, considering the myriad of potentially toxic substances to which an animal can be exposed, relatively few specific antidotes are commonly used in veterinary medicine. Because of the lack of specific antidotes, the veterinarian must treat each toxicity with general methods of poison management, applying basic critical care in the treatment of specific clinical signs associated with the poison exposure or toxicity. The adage "Treat the patient, not the poison" often comes into play when the exact toxic substance is unknown, or has no specific antidote.

ADVISING CLIENTS OVER THE PHONE

Before an animal arrives, the staff should be prepared to ask specific questions over the phone, and provide initial advice for clients, particularly if the animal lives some distance from the hospital (Box 1-55.)

TOXICOLOGY RESOURCES

It is important to have access to a database of information on toxic substances. Thousands of potentially toxic substances are available on the market today. The American Society for the Prevention of Cruelty to Animals (ASPCA) Animal Poison Control Center provides direct access to veterinary toxicologists 24 hours a day, 365 days a year. For additional information, call the nearest veterinary school or emergency center (Box 1-56). Also, see Section 6 for a table of emergency hotlines.

Human poison control centers

Check your local telephone book for a poison control center listing under Emergency numbers, usually found on the front cover. Although these numbers are for human poisonings, they have access to extensive poison and toxin databases and can potentially provide useful information for veterinarians, particularly regarding antidotal substances suitable for out of the ordinary toxins and human medications. Information on the toxic ingredients in thousands of medications, insecticides, pesticides, and other registered commercial products has been confidentially placed by the government in these poison control centers. As new products are marketed, information regarding toxin ingredients is forwarded to the centers.

Internet

Various e-mail discussion lists can serve as an informative resource for practitioners, but access generally requires an initial subscription and may have the disadvantage of delayed

BOX 1-55 TELEPHONE ADVICE FOR CLIENTS*

1. Questions to ask client:
 - Is your animal breathing or does it have respiratory difficulty? What is the color of the gums or tongue?
 - Is your animal able to walk?
 - Is there any vomiting, diarrhea, trembling, or seizures?
 - Does it appear lethargic or hyperactive?
 - What is the substance that your animal ingested (was exposed to)?
 - Did you witness the ingestion or exposure?
 - How much did the animal consume?
 - How long ago was the exposure?
 - Was the substance swallowed, or is it on the animal's skin or eyes?
 - How is the patient acting?
 - How long has the animal been acting that way?
- or
- When was the last time you saw your animal act normally?
2. First aid instructions for the client:
 - Induce vomiting at home and save the vomitus. Never induce vomiting if the patient is depressed, appears comatose, or is actively seizing. If the animal has ingested a caustic substance (strong alkali or acids) or a petroleum-based product (kerosene or turpentine), NEVER recommend induction of emesis.
 - Hydrogen peroxide (3% w/v[†])
 - 5 mL = 1 tsp/10 lb of body weight
 - Can repeat once if no vomiting occurs after 10 minutes
3. *Remind the owner* to bring a sample of the toxin and the vomitus in with the patient.
4. Advise the owner to *transport* the patient as rapidly as possible to the nearest veterinary hospital.

*Do not keep the client on the telephone for too long. Lengthy histories can be performed once the animal is at your hospital and you have started to initiate treatment.

[†]Hair dressing products sometimes have hydrogen peroxide as a 30% w/v; this concentration is not suitable for induction of emesis.

BOX 1-56 TOXICOLOGY DATABASES AND SUPPORTING RESOURCES**ASPCA ANIMAL POISON CONTROL CENTER**

1-888-426-4435 or 1-800-548-2423 (inside United States)

1-888-426-4435 (outside United States)

1717 S. Philo Road, Suite #36

Urbana, IL 61802

www.napcc.asPCA.org

A flat fee of \$50.00 US will be charged to a major credit card (Mastercard, Visa, Discover, or American Express).

Be ready to provide the following information:

Your name, address, and telephone number

Animal species, age, weight, gender, and number of animals involved

Information concerning exposure (what, how much, how long ago)

Clinical signs and onset of clinical signs after exposure

TEXTBOOKS

There are several excellent veterinary textbooks that provide detailed information on specific toxins:

Gfeller RW, Messonnier SP: *Handbook of small animal toxicology and poisoning*, St Louis, 1997, Mosby-Year Book; Veterinary Software Publishing, 1998.

Lorgue G, Lechenet J, Riviere A: *Clinical veterinary toxicology*, Cambridge, Mass, 1996, Blackwell Science.

Plumlee KH: *Clinical veterinary toxicology*, St Louis, 2003, Mosby.

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response times. They are useful for ideas on standard and long-term therapy, but not emergency stabilization. An exception to this is the Veterinary Interactive Network (VIN), which posts message board communications. Previous communications from veterinarians who treated a case with the same poison/toxin can be accessed with a subscription.

Manufacturers

Many manufacturers operate an information service about their products. If the product label or name is available, check for a telephone number that may route you to a specialist.

ESSENTIAL STEPS OF EMERGENCY TREATMENT OF TOXICITIES

There are six essential steps in treating toxicities:

1. Performing a physical examination
2. Stabilizing the patient's vital signs
3. Taking a thorough history
4. Preventing continued absorption of the toxin
5. Administering specific antidotes when available
6. Facilitating clearance or metabolism of the absorbed toxin

It is most important to provide symptomatic and supportive care both during and following emergency treatment.

The physical examination

Immediately on presentation, perform a brief but thorough physical examination. Obtain a minimum database as well as serum, urine, or orogastric lavage samples for later toxicologic analyses. It is important at this time to systematically evaluate the patient's physical status, focusing particularly on the toxins most common to a particular geographic location and the organ systems most commonly affected by toxins in veterinary medicine—namely, the neurologic and gastrointestinal tracts. A checklist is useful when performing a complete physical examination (Box 1-57).

Minimum database

The minimum database includes a urine sample, packed cell volume, total protein, serum urea, and serum glucose. The information obtained from these simple cage-side tests is useful for determining dehydration, hemoconcentration, azotemia (renal or pre-renal), and hypo- or hyperglycemia. When appropriate, obtain samples for serum biochemistry profiles, serum electrolytes, blood gases, serum osmolality, a complete hemogram, and coagulation profiles. Samples of serum, urine, and any vomitus or orogastric lavage contents should be collected and saved for later toxicologic analyses as required later.

Stabilization of vital signs

Stabilization of vital signs includes four major goals of treatment: maintain respiration, maintain cardiovascular function, control CNS excitation, and control body temperature. In any patient with clinical signs of respiratory distress or respiratory dysfunction, supplemental oxygen should be administered via flow-by, oxygen hood, oxygen cage, nasal, nasopharyngeal, or transtracheal oxygen sources. Ventilatory assistance may be necessary. Irritant or corrosive substances can cause damage to the oropharyngeal mucosa to such an extent that airway obstruction occurs. When necessary, a temporary tracheostomy should be performed. Arterial blood gases, pulse oximetry, and capnometry may be required to monitor oxygenation and ventilation.

At the time of presentation, immediately place an intravenous catheter for administration of intravenous fluids, inotropes, antiarrhythmics, and antidotes, if necessary. The initial fluid of choice is a balanced crystalloid solution such as Normosol-R, Plasmalyte-M, or lactated Ringer's solution. Fluid therapy can later be changed based on the patient's acid-base and electrolyte status. Some toxins can cause severe dysrhythmias and hyper- or hypotension. Monitor blood pressure and perform ECG and correct any abnormalities according to standard therapy (see sections on Hypotension and Cardiac Dysrhythmias).

EYES, EARS, NOSE, AND THROAT

What is the pupil size?

What is the pupil reactivity to light?

Is the ocular examination normal?

What is the sensitivity to light or sound?

Nose: Is it moist, dry, bubbling, or frothy, or caked with dirt?

Throat: Are there any characteristic odors on the breath?

Are there any traces of foreign material on the tongue or in the crevices of the teeth or gums?

Are there petechiae or ecchymosis on the gums or bleeding from the gumline?

CARDIOVASCULAR

What is the mucous membrane color? Is it normal and pink, or dark red (injected), pale, or icteric?

What is the capillary refill time? Is it fast, normal, or slow?

What is the patient's heart rate?

Are there any pulse deficits or dysrhythmias auscultated?

What is the patient's blood pressure?

What is the quality of the femoral pulse? Is it synchronous with the heart rate, or are there dropped pulses? Is the pulse bounding, normal, thready, or not palpable?

What is the patient's electrocardiogram?

RESPIRATORY

What is the patient's respiratory rate?

What is the patient's respiratory character? Is it normal, fast, shallow, or labored?

What do you hear on thoracic auscultation? Do you hear harsh airway sounds or pulmonary crackles?

GASTROINTESTINAL AND HEPATIC

What is the patient's rectal temperature?

Is there excessive salivation?

Is there evidence of vomiting or diarrhea?

Is abdominal palpation painful?

Do the intestinal loops feel normal, or are they fluid-filled or gas-filled?

What is the color and consistency of the feces?

UROGENITAL

Is there a palpable urinary bladder?

Is there urine production?

What is the color of the urine?

MUSCULOSKELETAL AND NEUROLOGIC

What is the patient's gait?

Is the patient weak or recumbent?

Is the patient ataxic?

Does the patient display signs of hypermetria?

Are there muscle fasciculations?

Is there increased extensor tone?

What is the patient's attitude?

Score the animal's level of consciousness on a simple scale:

Alert

Responds to voice

Responds to touch

Responds to pain/noxious stimulus

Unresponsive: unconscious

INTEGUMENT

Are there wet patches that smell of a particular substance?

Is there any evidence of erythema or ulcerations?

Does the muzzle, paws, prepuce, or vulva fluoresce with a black light?

PERIPHERAL LYMPH NODES

Peripheral lymph nodes should be normal in poisonings.

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Some toxins cause hemolysis, methemoglobinemia, Heinz body anemia, and coagulopathies. Whole blood, fresh frozen plasma, packed RBCs, or hemoglobin-based oxygen carriers should be available and used if necessary. Treat methemoglobinemia with a combination of ascorbic acid and *N*-acetylcysteine.

Many toxins affect the CNS, producing clinical signs of excitation and/or seizures. Diazepam is the drug of choice for most but not all seizures and tremors. If an animal has CNS excitation secondary to the ingestion of selective norepinephrine reuptake inhibitors, avoid using diazepam, as it can potentially exacerbate clinical signs. Muscle relaxants such as guaifenesin or methocarbamol may be required to control muscle spasm and tremors associated with some toxicities. Consider animals that are in status epilepticus because of toxin exposure at high risk. Such patients may not require the full dose of anesthetics or sedatives for seizure control. Give phenobarbital (16–20 mg/kg IV) or pentobarbital (3–25 mg/kg IV to effect) for longer-term management of seizures.

Core body temperature can easily increase or decrease secondary to increased muscle activity or coma. Animals may present as hypo- or hyperthermic, depending on the toxin ingested and the stage of toxicity. Manage hypothermia with circulating hot water or hot air blankets, or place bubble wrap or Saran wrap around the animal's peripheral extremities. Manage hyperthermia by placing lukewarm wet towels on the patient until the rectal temperature has decreased to 39.5° C (103° F). (See section on of Hyperthermia and Heat-induced Illness). If sedatives or anesthetics have been used, initial hyperthermia may initially resolve due to hypothalamic loss of thermoregulatory control, cool water bathing should not be performed.

Obtain a thorough history

When the patient is first presented to the veterinarian, have the owner complete a toxicologic history form (Figure 1-56) while the animal is being initially assessed and vital signs are being stabilized. When initial stabilization of vital signs has been accomplished, the veterinarian can discuss the patient's history with the owner. In urgent situations, the veterinarian should obtain a brief history as an initial procedure (Box 1-58).

Knowing when the animal was last seen as normal provides a time frame in which the toxic substance was most likely accessed, allowing differential diagnoses to be ranked in some order of probability by rate of onset. In eliciting a history from the owner about the animal's access to poisons, it is important not to take anything for granted. Many owners do not realize how poisonous some substances can be, such as insecticide products, garbage, cleaning chemicals, and over-the-counter drugs commonly used by humans. Many owners will deny that an animal could have ingested anything that might be toxic, not wanting to believe that the source of the toxin is within their household or property, particularly if recreational drug exposure is suspected. It is useful to phrase questions in a neutral fashion—for example, “Is such-and-such present on the premises?” rather than “Could the dog have eaten such-and-such?” If recreational drug exposure is suspected, another way to question the owners is to ask whether they have had any guests in their house recently that may have had such-and-such (e.g., marijuana, cocaine, methamphetamine). This approach serves to minimize the suggestion of any bias or preconceptions.

When questioning an owner about recent events, it is useful to realize and acknowledge that disruption in the household routine is a distinct factor in the occurrence accidents, including poisonings. Examples of such disruptive events include moving from the house, family member is ill or in the hospital, and renovations or recent construction. While these events are occurring, the safeguards followed by a normally careful owner may be disrupted. Often, doors or gates may be left open, animals may be outside instead of inside (or vice versa), and inexperienced people may be pet-sitters. Once owners are made aware of the importance of assessing such risks, they are often able to provide insight into otherwise baffling circumstances.

Prevent continued absorption of the toxin

Various methods can be used to remove toxins from the gastrointestinal tract, including emesis, orogastric lavage, cathartics, and enemas. Adsorbents, ion exchange resins, or

Toxicologic History Form**1****Date:****Time:****Patient information:****Name of animal:****Age:****Breed:****Gender and Neuter status:****Weight:****Vaccinations last given:****Any current medications (including heartworm prevention and nutraceuticals)****Today's Problem**

When did you first notice that something was wrong with your pet?

When was the last time you noticed your pet act normally?

What was the first abnormal sign noticed?

What other conditions have developed and what are they?

How soon did other signs develop?

Have the signs become better or worse since you first saw them?

Information on any suspected poison

What is the name of the product?

Do you have the container with you today?

Is it a liquid concentrate, dilute spray, or solid?

How long ago do you think that your pet was exposed to the poison?

Where do you think it happened?

Do you have any over-the-counter or prescription medications that your animal may have had access to?

Did you give any medications to your animal?

Is there any possibility of recreational drug exposure?

Your pet's recent activity

Did your pet eat this morning or last night?

What is he/she normally fed?

Is there a chance that your pet may have gotten into the garbage?

Have you fed table scraps or anything new recently? If so, what?

Has your pet been off your property in the last 24-48 hours?

Does your pet run loose unattended?

Has your pet had any antiflea/tick medication within the last week?

Your pet's environment

Is your animal kept inside or outside of the house?

Is your pet kept in a fenced-in yard or allowed to run loose unattended?

Does your pet have access to neighboring properties (even for a short time)?

Where has your pet been in the last 24 hours?

Has your pet traveled outside of your immediate geographic location? If so, when?

Has your pet been to rural areas in the last week?

Your household's recent activity

Has there been any gardening work recently?

Does your pet have access to a compost pile?

Any fertilizers or weed killer used in the last week?

Any construction work or renovation recently?

Any mouse or rat poison in your house, yard, or garage?

Any cleaning products used inside or outside the house within the last 48 hours? If so, which?

Have you changed your radiator fluid or does a car leak antifreeze?

Figure 1-56: Example of a thorough history form when a toxin is suspected.

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BOX 1-58 BRIEF HISTORY TO OBTAIN IN URGENT SITUATIONS

- When was the animal last seen as normal?
- What clinical signs developed?
- How fast did the clinical signs develop?
- When was the onset of clinical signs?
- What is the animal's activity level?
- Does the animal have access to any poisonous substances?
- This includes known toxins or chemicals, over-the-counter or prescription medications (including the owner's), and recreational drugs.

precipitating or chelating agents may be used. Removal of a toxic substance from the body surface may be necessary, depending on the toxin. The use of both emesis and orogastric lavage is less and less frequent in human medicine because of the risk of aspiration pneumonia and doubts about their efficacy. Currently, management of poisonings in human medicine relies heavily on the use of activated charcoal combined with sorbitol as a cathartic, when appropriate, and supportive critical care. It should be emphasized, however, that the majority of poisonings in humans are due to drug overdoses (illicit or otherwise) (which have a relatively small volume and rapid absorption), for which this treatment is appropriate. Furthermore, adoption of the approach rests on the availability of a hospital intensive care infrastructure, which is not always available in veterinary practice.

Emetics

Induce emesis if the animal's physiology and neurologic status are stable (i.e., does not have respiratory depression or is not actively seizing, obtunded, unable to swallow or protect its airway). Do not administer the same emetic more than twice. If the emetic doesn't work after two doses, give a different emetic or perform orogastric lavage under general anesthesia. *Emetics are strictly contraindicated for toxicity from petroleum-based products and corrosives because of the risk of aspiration pneumonia and further esophageal damage.* Emetics may also be of little value if poisons with antiemetic properties have been ingested, such as benzodiazepines, tricyclic antidepressants, and marijuana (Table 1-50).

Various emetics traditionally have been recommended for use in veterinary medicine. Many have fallen out of favor because of the risk of causing adverse consequences and side effects. Apomorphine (0.04 mg/kg IV or in the conjunctival sac) remains the standard but is less useful in certain situations in which the poison causes CNS excitation or stimulation. It is ineffective in cats. Other emetics include xylazine and hydrogen peroxide. Do NOT use table salt because of the risk of severe oropharyngeal irritation and hypernatremia. Do not use mustard powder or dishwashing liquid detergent because of the risk of severe oropharyngeal, esophageal, and gastric irritation.

Orogastric lavage

Orogastric lavage is described in detail in the section on Emergency Procedures. Gastric lavage is contraindicated in treatment of toxicity from petroleum-based compounds and acid/alkali ingestion. The procedure can be messy but is very effective if performed within 1 to 2 hours of ingestion of the poison. To prevent aspiration, the patient should be placed under general anesthesia. Keep the animal's head lowered during the procedure to prevent aspiration of stomach contents into the trachea. It is sometimes helpful to put the animal in both right and left lateral recumbency to allow complete emptying of gastric contents. Repeat the procedure until the fluid runs clear from the stomach. In some cases in which solid material has been ingested, this process can take a long time, so be prepared with a large volume of warm water.

Following successful evacuation and lavage, administer a slurry of activated charcoal through the orogastric tube before removing it. Keep the endotracheal tube cuffed and in place until the animal is semi-conscious, is starting the fight the tube, and is visibly able to swallow and protect its airway.

TABLE 1-50 List of Emetics and Recommended Doses

Name	Mechanism of action	Dose/onset	How supplied	Adverse effects
Apomorphine	Dopaminergic-receptor stimulation in chemoreceptor trigger zone; causes both central nervous system (CNS) depression and stimulation and some respiratory depression	0.02-0.04 mg/kg IV or in conjunctival sac	6.25-mg tablets, can be compounded into sterile capsules for intravenous use	Respiratory and CNS depression Undesirable CNS excitement in metaldehyde toxicosis
Hydrogen peroxide	Gastric irritation	1-2 mL/kg	3% solution PO, can be repeated once every 10 minutes	Protracted vomiting; some formulations have a stabilizing factor that can be converted into acetaminophen; use caution in very small dogs and in cats
Xylazine	Central α 2-agonist stimulation	0.5-1.0 mg/kg IM every 10-15 minutes	Solution	Sedation, bradycardia, respiratory depression

BOX 1-59 EQUIPMENT NEEDED FOR ENEMA ADMINISTRATION**TUBING**

Flexible red rubber catheters

Foley balloon-tipped catheters if a retention enema is required

OBSTETRIC LUBRICANT

Use nonsterile nonspermicidal water-soluble lubricants (K-Y jelly)

FLUID RESERVOIR

Old intravenous fluid bag

Enema bag

60- to 120-mL syringe

FLUID

Warm water, with or without hand or liquid dish soap

Enemas

Enemas are useful to facilitate the action of cathartics and in cases in which the poison is a solid material (e.g., compost, snail bait, garbage) (Box 1-59). It is best to use just lukewarm water. Commercially available phosphate enema solutions can cause severe electrolyte disturbances (hyperphosphatemia, hyponatremia, hypocalcemia, and hypomagnesemia) and acid-base abnormalities (metabolic acidosis); therefore, they are absolutely contraindicated in small animal patients.

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The fluid volume required depends on the size of the animal and the state of its lower gastrointestinal tract. As with orogastric lavage, continue the procedure until the water runs clear. If difficulty is encountered emptying the lower gastrointestinal tract, repeat the enema in 1 or 2 hours, rather than be overzealous on the first attempt.

Cathartics

Cathartics are useful for hastening gastrointestinal elimination of toxins, and they are particularly useful for elimination of most solid toxicants (e.g., compost, garbage, snail baits). Cathartics can be used in conjunction with activated charcoal. Do not use magnesium-based cathartics in patients with CNS depression, because hypermagnesemia can worsen this disorder and also cause cardiac rhythm disturbances (Table 1-51).

Activated charcoal (1-4 mL/kg) is the safest and to date the most effective adsorbent for the treatment of ingested toxins. Activated charcoal can be administered after emesis or orogastric lavage or can be administered as the sole treatment. Various preparations are available on the market, including dry powder, compressed tablets, granules, liquid suspensions, and concentrated paste preparations. Commercially available products are relatively inexpensive and should be used whenever possible for ease of administration. Vegetable-origin activated charcoal is the most efficient adsorbent and binds compounds with weak, nonionic bonds. Some preparations are combined with sorbitol to provide simultaneous administration of an adsorbent and a cathartic; this combination has been shown to be most efficacious.

Repeated administration of activated charcoal every 4 to 6 hours has been shown to be beneficial in the management of a toxin that undergoes enterohepatic recirculation. Administration of an oily cathartic or mixing the activated charcoal with food only serves to reduce the absorptive surface of the activated charcoal and therefore is not recommended. In general, substances that are very soluble and are rapidly absorbed are not well adsorbed by activated charcoal, including alkalis, nitrates, mineral acids, ethanol, methanol, ferrous sulfate, ammonia, and cyanide.

Kaolin and bentonite are clays that have been used as adsorbents. Both are usually less effective than activated charcoal. However, they are reported to be better adsorbents than activated charcoal for the herbicide paraquat.

Ion exchange resins

Ion exchange resins can ionically bind certain drugs or toxins. Cholestyramine is one such resin, commonly used in human medicine to bind intestinal bile acids and thereby decrease cholesterol absorption. Its application in toxicology extends to the absorption of

TABLE 1 - 51 List of Cathartics and Recommended Doses

Product*	Dose
Sodium sulfate (Glauber's salts, GoLYTELY)	250-500 mg/kg PO in 10 times volume of water
Sorbitol (70%)	3 mL/kg PO
Mineral oil (paraffin oil)	5-15 mL per dog; 2-6 mL/cat Is not normally absorbed across the intestinal wall; do not use along with dioctyl sodium sulfosuccinate because emulsification can cause accumulation of indigestible oil in the liver; is no longer recommended for organophosphate insecticide and other organic compound ingestions

*Vegetable oil and Epsom salts (magnesium sulfate) are no longer recommended.
Note: Administer all cathartics at least 30 minutes after activated charcoal. Many activated charcoal products have sorbitol or other cathartic with the activated charcoal, so administration of a different cathartic is often unnecessary.

fat-soluble toxins such as organochlorine and certain acidic compounds such as digitalis. Ion exchange resins also have been used to delay or reduce the absorption of phenylbutazone, warfarin, chlorothiazide, tetracycline, phenobarbital, and thyroid preparations.

Precipitating, chelating, and diluting agents

Precipitating, chelating, and diluting agents are used primarily in the management of heavy metal intoxications, such as alkaloids or oxalates. They work by binding preferentially to the metal ion and creating a more soluble complex that is amenable to renal excretion. Those chelating agents in common usage are calcium EDTA, deferoxamine, and D-penicillamine. Calcium EDTA and deferoxamine should both be on hand in the veterinary hospital because they are necessary to treat zinc and iron toxicity, respectively, both of which have a short window of opportunity for therapeutic intervention. D-Penicillamine has a wide application for a number of metal toxicities but tends to be used for long-term chronic therapy because it can be administered orally. Various agents used for nonspecific dilution of toxins, including Milk of Magnesia and egg whites, although old-fashioned, still have wide application in many cases in which low-grade irritants have been ingested.

Eliminating poison from the skin

Bathing the animal is an important aspect of treatment for topical exposures to toxins such as insecticidal products, petroleum-based products, and aromatic oils. Bathing an animal is not an innocuous procedure. To avoid hypothermia and shock, use warm water at all times. Actively dry the animal to further minimize the risk of hypothermia. When bathing the animal, use rubber gloves and a plastic apron to avoid exposure to noxious agents.

In most cases, a mild dishwashing soap is appropriate. Medicated or antibacterial shampoos are less appropriate in this situation. For petroleum-based products in particular, Dawn dishwashing liquid that “cuts the grease” works well to remove the oils. If Dawn is not available, mechanics’ hand cleaners or coconut oil-based soaps can be used instead. As a general principle, best results are obtained by barely wetting the patient’s fur until the detergent is worked well into the fur, keeping the amount of water to a minimum until ready for the rinse. Oil-based paint is best removed by clipping rather than by attempting removal with solvents, because solvents are also toxic.

To remove powder products, brush and vacuum the animal before bathing it to eliminate further toxic exposure. With caustic alkaline or acidic products, the primary treatment is to dilute and flush the skin with warm water; do NOT attempt neutralization. Neutralization can cause an exothermic reaction that causes further damage to the underlying tissues.

Eliminating poison from the eyes

For ocular exposures, irrigate the eyes for a minimum of 20 to 30 minutes with warm (body temperature) tap water or warmed 0.9% sterile saline solution. The use of neutralizing substances is not recommended because of the risk of causing further ocular damage. Following adequate irrigation, treat chemical burns of the eyes with lubricating ointments and possibly a temporary tarsorrhaphy. Atropine may be indicated as a cycloplegic agent. Systemic nonsteroidal antiinflammatory drugs can be used to control patient discomfort.

Daily follow-up examinations are required because epithelial damage may be delayed, especially with alkali burns, and it is difficult to predict the final extent of ocular damage. Topical glucocorticosteroids are contraindicated if the corneal epithelium is not intact. If severe conjunctival swelling is present with a corneal ulcer, parenteral glucocorticosteroids can be administered to help alleviate inflammation, but nonsteroidal antiinflammatory drugs should not be used simultaneously due to the risk of gastrointestinal ulceration or perforation.

Administer antidotes

Whenever possible, administer specific antidotes to negate the effects of the toxin and prevent conversion of the substance to the toxic metabolite. Three categories of agents are used in the management of poisonings.

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The first category is specific antidotes. Unfortunately, few specific antidotes are available for use in veterinary medicine. Some “classic” toxins and antidotes are now considered to be rare, such as curare and physostigmine, thallium and Prussian blue, and fluoride and calcium borogluconate. These and a few others have been omitted from the table.

The second, broader category of antidotes includes those drugs used in the symptomatic management of clinical signs, which are part of our routine veterinary stock. Drugs such as atropine, sedatives, steroids, antiarrhythmics, and beta-blockers fall into this category.

The third category comprises nonspecific decontaminants such as activated charcoal, cathartics, and emetics. These were discussed previously.

Facilitate clearance or metabolism of absorbed toxin

Many patients benefit from efforts to enhance clearance or metabolism of the absorbed toxins. Some specific therapies have been developed for this purpose, including 4-methylpyrazole for ethylene glycol toxicity and specific antibodies such as Digibind (digoxin immune Fab [ovine]) for digitalis toxicity. Other strategies are aimed at promoting renal excretion. Renal excretion strategies include diuresis, ion trapping, and peritoneal dialysis or hemodialysis (see section on Peritoneal Dialysis). Diuresis and ion trapping are applicable to a large number of toxins and are discussed here in more detail. Other toxins respond to urine acidification and urine alkalinization.

Enhancing renal excretion of substances is most useful for those organic substances that are present in significant concentrations in the plasma. Substances that are non-ionic and lipid-soluble, such as certain herbicides, are likely to be less affected by attempts to promote rapid renal elimination.

Before starting diuresis or ion trapping, intravenous fluid therapy should be adequate as determined by normal central venous pressure, urine output, and mean arterial blood pressure. If any of these values are less than normal, use other measures to ensure adequate renal perfusion, including but not limited to a constant rate infusion of dopamine.

Simple fluid diuresis can influence the excretion of certain substances. The use of mannitol as an osmotic diuretic may reduce the passive reabsorption of some toxic substances in the proximal renal convoluted tubule by reducing water reabsorption. Dextrose (50%) can be used as an osmotic diuretic. Furosemide can be used to promote diuresis, but again, there is no substitute for intravenous fluid therapy. The use of mannitol, dextrose, and furosemide is contraindicated in hypotensive or hypovolemic patients. Take care to avoid causing dehydration with any diuretic; central venous pressure monitoring is strongly recommended.

Urine acidification and alkalinization

Ion trapping is based on the principle that ionized substances do not cross renal tubular membranes easily, and are not well reabsorbed. If the urinary pH can be changed so that the toxin's chemical equilibrium shifts to its ionized form, then that toxin can be “trapped” in the urine and excreted. Alkaline urine favors the ionization of acidic compounds, and acidic urine favors the ionization of alkaline compounds. Those toxins that are amenable to ion trapping are mostly weak acids and weak bases.

Ammonium chloride can be used to promote urinary acidification. Contraindications to the use of ammonium chloride include a preexisting metabolic acidosis, hepatic or renal insufficiency, and hemolysis or rhabdomyolysis leading to hemoglobinuria or myoglobinuria. Signs of ammonia intoxication include CNS depression and coma. When performing urine acidification, frequently check the serum potassium concentration and urine pH.

Urine alkalinization can be performed with use of sodium bicarbonate. Contraindications to the use of sodium bicarbonate include metabolic alkalosis (particularly with concurrent use of furosemide), hypocalcemia, and hypokalemia. As with urine acidification, monitor the serum potassium concentration and urine pH frequently.

SUPPORTIVE AND SYMPTOMATIC CARE OF THE POISONING PATIENT

The major steps in management of poisonings discussed here must be accompanied by application of the fundamentals of critical care. Respiratory and cardiovascular support have been discussed previously. Renal and gastrointestinal function and analgesia are particularly important in the management of the poisoning patient.

Maintenance of renal perfusion is a priority in the poisoning patient. Fluid, electrolyte, and acid-base balance must be controlled and be accurate. Poisoning patients are at particularly high risk for renal damage and acute renal failure, whether by primary toxic insult to the renal parenchyma or by acute or prolonged renal hypoperfusion. For this reason, a protocol that aims at preventing oliguria and ensuing renal failure is one of the therapeutic strategies that should be routinely employed. This protocol is described in Box 1-60.

Gastrointestinal protectants

Gastrointestinal protectant drugs may be indicated for the management of those poisons that are gastrointestinal irritants or ulcerogenic. Commonly used gastroprotectant drugs include cimetidine, ranitidine, famotidine, omeprazole, sucralfate, and misoprostol.

Antiemetics

Antiemetics may be used to suppress intractable vomiting. Metoclopramide is commonly used, and it is the drug of choice for centrally mediated nausea. Antiemetics that work by different mechanisms can be used in combination as necessary. Examples are dopamine 2-receptor antagonists such as prochlorperazine, 5-hydroxytryptamine antagonists such as ondansetron and dolasetron, and H-1 receptor antagonists such as diphenhydramine and meclizine.

Analgesics

Analgesics are more appropriate to treat poisonings than once thought. Common effects of poisons including severe gastroenteritis and topical burns or ulcerations may warrant the use of analgesics. Longer-acting analgesics such as morphine, hydromorphone, and buprenorphine are particularly useful.

Nutritional support

Nutritional support may be necessary in the form of enteral or parenteral feeding in patients that have esophageal or gastric damage or that need to be sedated for long periods of time. Endoscopy may be useful in assessing the degree of esophageal and gastric damage, particularly after ingestion of caustic substances.

BOX 1-60 MAINTENANCE OF RENAL PERFUSION

1. Administer crystalloid intravenous fluids at maintenance rates using a balanced electrolyte solution.
2. Perform urinary catheterization and collection to monitor urine output.
3. Monitor serum urea nitrogen and creatinine every 12 hours.
4. Monitor serum electrolytes every 6 to 8 hours.
5. Monitor central venous pressure every 2 to 4 hours.
6. Treat oliguria, defined as a drop in urine output to less than 1 mL/kg/hour.
7. Initiate a fluid challenge with a crystalloid or colloid (5 mL/kg) bolus.
8. Start dopamine at 3 to 5 μ g/kg/minute if no response to crystalloid/colloid bolus occurs within 30 minutes.
- 9a. Consider mannitol (0.5 to 1 g/kg IV) administration if no response to dopamine occurs within 30 minutes.
- 9b. Consider furosemide (4 to 8 mg/kg IV, or 0.66 to 1 mg/kg/hour IV CRI) if no response to dopamine or mannitol occurs in 30 to 60 minutes.
10. If no response to furosemide, peritoneal dialysis or hemodialysis is indicated immediately, particularly if anuria is present.

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TREATMENT OF SPECIFIC TOXINS**Acetaminophen (paracetamol)***Introduction:*

Acetaminophen (paracetamol) is the active ingredient in Tylenol and many over-the-counter cold products. Acetaminophen is converted to *N*-acetyl-P-benzoquinonimine in the liver, a toxic substance that can cause oxidative injury of red blood cells and hepatocytes. Clinical signs of acetaminophen toxicity include respiratory distress from lack of oxygen-carrying capacity, cyanosis, methemoglobinemia (chocolate-brown appearance of the blood and mucous membranes), lethargy, vomiting, and facial and paw swelling (cats). The toxic dose of acetaminophen is >100 mg/kg for dogs, and 50 mg/kg for cats.

Treatment:

Treatment of acetaminophen toxicity includes induction of emesis or orogastric lavage if the substance has been ingested within 30 minutes. Activated charcoal should also be administered. In cases of severe anemia, give supplemental oxygen along with a packed RBC transfusion. Administer intravenous fluids to maintain renal and hepatic perfusion. *N*-acetylcysteine, vitamin C, and cimetidine are the treatments of choice for methemoglobinemia in patients with acetaminophen toxicity.

Acids/corrosives*Introduction:*

Hydrochloric, nitric, and phosphoric acids cause chemical burns through contact with the skin and/or eyes. Localized superficial coagulative necrosis occurs upon contact. Usually, the patient's skin is painful to the touch or the animal may lick or chew at an irritated area that is not visible under the haircoat.

Treatment:

If the chemical is swallowed, do NOT induce emesis or perform orogastric lavage, because of the risk of worsening esophageal irritation. Rinse the patient's skin and eyes with warm water or warm saline for a minimum of ½ hour. Use analgesics and treat corneal ulcers (see section on Corneal Ulcers) as required. Do not attempt chemical neutralization, because of the risk of causing an exothermic reaction and worsening tissue injury.

Aflatoxin*Introduction:*

Aflatoxin (*Aspergillus flavus*) is found in moldy feed grains. Clinical signs of toxicity occur after ingestion and include vomiting, diarrhea, and acute hepatitis; abortion may occur in pregnant bitches.

Treatment:

Treatment of suspected aflatoxin ingestion consists of gastric decontamination, administration of activated charcoal, intravenous fluids, and hepatic supportive care (S-Adenosyl Methionine [SAME], milk thistle).

Alcohols*Introduction:*

Drinking (ethanol), rubbing (isopropyl), and methyl (methanol) alcohols can be harmful if ingested (4.1 to 8.0 g/kg PO). All cause disruption of neuronal membrane structure, impaired motor coordination, CNS excitation followed by depression, and stupor that can lead to cardiac and respiratory arrest, depending on the amount ingested. Affected animals may appear excited and then ataxic and lethargic. Contact or inhalant injury can occur, causing dermal irritation and cutaneous hyperemia. Methanol also can cause hepatotoxicity.

Treatment:

Induce and maintain a patent airway and stabilize the patient's cardiovascular and respiratory status. Control CNS excitation with diazepam, if necessary, and control the patient's body temperature (both hypo- and hyperthermia). Induce vomiting if the patient is alert and can protect its airway; otherwise, perform orogastric lavage with the patient under general anesthesia with a cuffed endotracheal tube in place. Alcohols do not bind well with activated charcoal. Treat dermal exposure by bathing the area with warm water.

Alkalis/caustics*Introduction:*

If ingested, sodium or potassium hydroxide can cause severe contact dermatitis or irritation of the gastrointestinal tract. Esophageal burns and full-thickness coagulative necrosis can occur.

Treatment:

If an animal ingests a caustic alkali substance, feed the animal four egg whites mixed with 1 quart of warmed water. Perform endoscopy within 24 hours to evaluate the extent of injury and to place a feeding tube, in severe cases. Do NOT induce emesis, and do NOT perform orogastric lavage, because of the risk of worsening esophageal irritation. In cases of contact exposure to the skin or eyes, rinse the exposed area with warm water baths for at least 30 minutes. Administer gastroprotectant, antiemetic, and analgesic drugs as necessary. Avoid neutralization, which can cause a hyperthermic reaction and worsen injury to the skin and gastrointestinal tract.

Amitraz*Introduction:*

Amitraz is the active ingredient in ascaricides and anti-tick and anti-mite products such as Mitaban and Taktic. The toxic dose is 10 to 20 mg/kg. Amitraz exerts its toxic effects by causing α -adrenergic stimulation, and causes clinical signs similar to those observed with administration of xylazine: bradycardia, CNS depression, ataxia, hypotension, hyperglycemia, hypothermia, cyanotic mucous membranes, polyuria, mydriasis, and emesis. A coma can develop.

Treatment:

Treatment of amitraz intoxication includes cardiovascular support with intravenous crystalloid fluids and induction of emesis in asymptomatic animals. If clinical signs are present, orogastric lavage may be required. Many toxic compounds are impregnated in a collar form. If the patient has ingested a collar and does not vomit it, it should be removed using endoscopy or gastrotomy. Administer activated charcoal to prevent or delay absorption of the toxic compound. Yohimbine or atepamizole, both α -adrenergic antagonists, are the treatment(s) of choice to reverse the clinical signs of toxicity. Avoid the use of atropine, because it can potentially increase the viscosity of respiratory secretions and cause gastrointestinal ileus, thus promoting increased absorption of the toxic compound.

Ammonia, cleaning*Introduction*

Ammonium hydroxide, or cleaning ammonia, can be caustic at high concentrations (see Alkalis/Caustics) and cause severe injury to the respiratory system if inhaled. Pulmonary edema or pneumonia can occur, resulting in respiratory distress. Ingestion of ammonia can cause severe irritation to the gastrointestinal tract and cause vomiting and esophageal injury.

Treatment:

If ammonia is ingested, administer a dilute solution of egg white.

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Administer gastroprotectant, antiemetic, and analgesic drugs as necessary. If pneumonia or pulmonary edema occurs secondary to aspiration of ammonia into the airways and alveolar spaces, treatment is largely supportive with supplemental oxygen administration, antibiotics, fluid therapy, and mechanical ventilation as necessary. Diuretics may or may not be useful in the treatment of pulmonary edema secondary to ammonia inhalation.

Amphetamine*Introduction*

Amphetamines cause CNS excitation due to neurosynaptic stimulation, resulting in hypersensitivity to noise and motion, agitation, tremors, vomiting, diarrhea, and seizures. Clinical signs of amphetamine toxicity include muscle tremors, tachyarrhythmias, mydriasis, ptialism, and hyperthermia.

Treatment

Amphetamines are rapidly absorbed from the gastrointestinal tract. Treatment includes administration of intravenous fluids to maintain hydration and renal perfusion and correction of hyperthermia. Administer sedative drugs such as chlorpromazine to control agitation and tremors, and diazepam to control seizures. Urinary acidification can promote excretion and prevent reabsorption from the urinary bladder. In severe cases, treat cerebral edema with a combination of mannitol followed by furosemide to control increased intracranial pressure.

Antifreeze: see ethylene glycol**Antihistamines***Introduction*

Antihistamines (loratadine, diphenhydramine, doxylamine, clemastine, meclizine, dimenhydrinate, chlorpheniramine, cyclizine, terfenadine, hydroxyzine) are available as over-the-counter and prescription allergy and anti-motion sickness products. Clinical signs of antihistamine toxicity include restlessness, nausea, vomiting, agitation, seizures, hyperthermia, and tachyarrhythmias.

Treatment

Treatment of antihistamine intoxication is largely symptomatic and supportive, as there is no known antidote. If ingestion is recent (within 1 to 2 hours) and the patient is not actively seizing and can protect its airway, induce emesis or perform orogastric lavage, followed by administration of activated charcoal and a cathartic. Monitor the patient's heart rate, rhythm, and blood pressure. Treat cardiac arrhythmias, if present, with appropriate therapies (see section on Cardiac Dysrhythmias). Administer cooling measures and intravenous fluids to treat hyperthermia. A constant rate infusion of guaifenesin can be used to control muscle tremors.

ANTU (α -naphthylthiourea)*Introduction*

α -Naphthylthiourea (ANTU) is manufactured as a white or blue-gray powder. The toxic dose in dogs is 10-40 mg/kg, and in cats is 75-100 mg/kg. Younger dogs appear to be more resistant to its toxic effects. ANTU usually causes profound emesis and increased capillary permeability that eventually leads to pulmonary edema.

Treatment

Treatment of ANTU toxicity includes respiratory support. Mechanical ventilation may be required in severe cases of pulmonary edema. If an animal does not vomit, orogastric lavage should be performed. Administer gastrointestinal protectant, antiemetic, and analgesic drugs. Cardiovascular support in the form of intravenous crystalloids should be

administered with caution, because of the risk of exacerbating increased capillary permeability and causing pulmonary edema.

Arsenic

Introduction

Inorganic arsenic (arsenic trioxide, sodium arsenite, sodium arsenate) is the active ingredient in many herbicides, defoliants, and insecticides, including ant killers. The toxic dose of sodium arsenate is 100-150 mg/kg; that of sodium arsenite is 1-25 mg/kg. Sodium arsenite is less toxic, although cats are very susceptible. Arsenic compounds interfere with cellular respiration by combining with sulfhydryl enzymes. Clinical signs of toxicity include severe gastroenteritis, muscle weakness, capillary damage, hypotension, renal failure, seizures, and death. In many cases, clinical signs are acute in onset.

Treatment

Treatment of arsenic toxicity involves procuring and maintaining a patent airway. Administer intravenous crystalloid fluids to correct hypotension and hypovolemia, and normalize acid-base and electrolyte balance. If no clinical signs are present and if the compound was ingested within 2 hours, induce emesis. If clinical signs are present, perform orogastric lavage followed by administration of activated charcoal. If dermal exposure has occurred, thoroughly bathe the animal to prevent further absorption. Dimercaprol (BAL, 3-4 mg/kg IM q8h) can be administered as a chelating agent. *N*-acetylcysteine (Mucomyst) (for cats, 140-240 mg/kg PO IV, then 70 mg/kg PO IV q6h for 3 days; for dogs, 280 mg/kg PO or IV, then 140 mg/kg PO IV q4h for 3 days) has been shown to decrease arsenic toxicity in rats.

Aspirin (acetylsalicylic acid, salicylate)

Introduction

Aspirin causes inhibition of the production of prostaglandins, a high anion gap metabolic acidosis, gastrointestinal ulceration, hypophosphatemia, and decreased platelet aggregation when ingested in high quantities (>50 mg/kg/24 hours in dogs; >25 mg/kg/24 hours in cats). Clinical signs of aspirin toxicity include tachypnea, vomiting, anorexia, lethargy, hematemesis, and melena.

Treatment

Treatment of aspirin toxicity is largely supportive. If the ingestion was recent (within the last hour), induce emesis or perform orogastric lavage followed by administration of activated charcoal. Administer intravenous crystalloid fluids to maintain hydration and correct acid-base abnormalities. Administer synthetic prostaglandin analogues (misoprostol), gastroprotectant drugs, and antiemetics. Alkalinization of the urine can enhance excretion.

Atomoxetine: see Strattera

Baclofen

Introduction

Baclofen is a GABA agonist centrally acting muscle relaxant. Clinical signs of toxicity include vomiting, ataxia, vocalization, disorientation, seizures, hypoventilation, coma, and apnea. Clinical signs can occur at doses as low as 1.3 mg/kg.

Treatment

Treatment of baclofen ingestion includes induction of emesis if the animal is asymptomatic. Otherwise, perform orogastric lavage. Emesis or orogastric lavage should be followed by administration of activated charcoal. Perform intravenous crystalloid fluid diuresis to promote elimination of the toxin, maintain renal perfusion, and normalize body temperature. Supplemental oxygen or mechanical ventilation may be required for hypoventilation or apnea. If seizures occur, avoid the use of diazepam, which is a GABA agonist and can potentially worsen clinical signs. Control seizures with intravenous

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phenobarbital, pentobarbital, or propofol. Supportive care (eye lubrication, urinary catheter placement for patient cleanliness, passive range of motion exercises, soft heavy bedding to prevent decubitus ulcer formation) is required. Clinical signs usually resolve in several days. Seizures warrant a more guarded prognosis.

 β -Adrenergic agonists (asthma inhalers/medications)*Introduction*

β -adrenergic agonists, including terbutaline, albuterol (salbutamol), and metaproterenol, are commonly used in inhaled form for the treatment of asthma. Animals commonly are exposed to the compounds after chewing on their owners' inhalers. Clinical signs of β -adrenergic stimulation include tachycardia, muscle tremors, and agitation. Severe hypokalemia can occur.

Treatment

Treatment of β -adrenergic agonist intoxication includes treatment with beta-blockers (propranolol, esmolol, Atenolol), intravenous fluids, and intravenous potassium supplementation. Diazepam or acepromazine may be administered for sedation and muscle relaxation.

Barbecue lighter fluids: see fuels**Barbiturates***Introduction*

Barbiturates such as phenobarbital are GABA agonists and induce CNS depression. Clinical signs of barbiturate overdose or toxicity include weakness, lethargy, hypotension, hypoventilation, stupor, coma, and death.

Treatment

Treatment of barbiturate toxicity includes maintenance and support of the cardiovascular and respiratory systems. If clinical signs are absent and the patient can protect its airway, induce emesis followed by repeated doses of activated charcoal. Perform orogastric lavage if emesis is contraindicated. Administer supplemental oxygen if hypoventilation occurs. Some animals may require mechanical ventilation. Administer intravenous fluids to control perfusion and blood pressure. Positive inotropic drugs may be required if dose-dependent decrease in cardiac output and blood pressure occurs. Alkalinization of the urine and peritoneal dialysis can be performed to enhance excretion and elimination. Hemodialysis should be considered in severe cases, if available.

Batteries*Introduction*

Automotive and dry cell batteries contain sulfuric acid that can be irritating on contact with the eyes, skin, and gastrointestinal tract. Button batteries, which contain sodium or potassium hydroxide, cause contact irritation if chewed.

Treatment

To treat exposure, rinse the eyes and skin with copious amounts of warm tap water or sterile saline solution for a minimum of 30 minutes. If ingestion occurred, administer gastro-protectant and antiemetic drugs. Induction of emesis and orogastric lavage is absolutely contraindicated because of the risk of aspiration pneumonia and worsening esophageal irritation. No attempt should be made at performing neutralization because of the risk of causing an exothermic reaction and worsening tissue damage. Administer analgesics to control discomfort.

Benzoyl peroxide*Introduction*

Benzoyl peroxide is the active ingredient in many over-the-counter acne preparations. Ingestion can result in production of hydrogen peroxide, gastroenteritis, and gastric dilatation. Topical exposure can cause dermal irritation and blistering.

Treatment

If an animal has ingested benzoyl peroxide, do NOT induce emesis, because of the risk of worsening esophageal irritation. Instead, perform orogastric lavage. Administer gastroprotectant and antiemetic medications and closely observe the patient observed for signs of gastric dilatation.

Bismuth subsalicylate (Pepto-Bismol): see aspirin

Bleach, chlorine (sodium hypochlorite)

Introduction

Sodium hypochlorite is available in dilute (3%-6%) or concentrated (50% industrial strength or swimming pool) solutions for a variety of purposes. Sodium hypochlorite can cause severe contact irritation and tissue destruction, depending on the concentration. Affected animals may have a bleached haircoat.

Treatment

Treatment of exposure includes dilution with copious amounts of warm water or saline baths and ocular lavage. Induction of emesis and orogastric lavage is absolutely contraindicated because of the risk of causing further esophageal irritation. To treat ingestion, give the animal milk or large amounts of water, in combination with gastroprotectant and antiemetic drugs, to dilute the contents in the stomach. Administration of sodium bicarbonate or Milk of Magnesia is no longer recommended.

Bleach, nonchlorine

Introduction

Nonchlorine bleaches (sodium peroxide or sodium perborate) have a moderate toxic potential if ingested. Sodium peroxide can cause gastric distention. Sodium perborate can cause severe gastric irritation, with vomiting and diarrhea; renal damage and CNS excitation followed by depression can occur, depending on the amount ingested.

Treatment

To treat dermal or ocular exposure, rinse the skin or eyes with copious amounts of warm tap water or sterile saline for a minimum of 30 minutes; treat ocular injuries as necessary, if corneal burns have occurred. If the bleach has been ingested, DO induce emesis and perform orogastric lavage. Administer Milk of Magnesia (2-3 mL/kg).

Boric acid, borate

Introduction

Boric acid is the active ingredient in many ant and roach killers. The toxic ingredient (in amounts of 1-3 g/kg) can cause clinical signs in dogs by an unknown mechanism. Clinical signs include vomiting (blue-green vomitus), blue-green stools, renal damage, and CNS excitation and depression.

Treatment

Treatment of boric acid or borate ingestion includes gastric decontamination with induction of emesis or orogastric lavage, followed by administration of a cathartic to hasten elimination. Activated charcoal is not useful to treat ingestion of this toxin. Administer intravenous fluid therapy to maintain renal perfusion. Administer gastroprotectant and antiemetic drugs, as necessary.

Botulism

Introduction

Clostridium botulinum endospores can be found in carrion, food, garbage, and the environment. Ingestion of endospores and *C. botulinum* endotoxin rarely can cause generalized neuromuscular blockade of spinal and cranial nerves, resulting in miosis, anisocoria, lower motor neuron weakness, and paralysis. Respiratory paralysis, megaesophagus, and aspiration pneumonia can occur. Clinical signs usually develop within 6 days of ingestion.

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Differential diagnosis includes acute polyradiculoneuritis (coonhound paralysis), bromethalin intoxication, and tick paralysis.

Treatment

Treatment of botulism is largely supportive; although an antitoxin exists, it often is of no benefit. Treatment may include administration of intravenous fluids, frequent turning of the patient and passive range-of-motion exercises to prevent disuse muscle atrophy, and supplemental oxygen administration or mechanical ventilation. Administer amoxicillin, ampicillin, or metronidazole. Recovery may be prolonged, up to 3 to 4 weeks in some cases.

Bromethalin*Introduction*

Bromethalin is the active ingredient in some brands of mouse and rat poisons. It usually is packaged as 0.01% bromethalin in green or tan pellets, and packaged in 16 – 42.5 g place packs. The toxic dose for dogs is 116.7 g/kg, and for cats 3 g/kg. Bromethalin causes toxicity by uncoupling of oxidative phosphorylation. An acute syndrome of vomiting, tremors, extensor rigidity, and seizures occurs within 24 hours of ingestion of high doses. Delayed clinical signs occur within 3 to 7 days of ingestion of a lower dose and include posterior paresis progressing to ascending paralysis, CNS depression, and coma.

Treatment

Treatment of known bromethalin ingestion includes induction of emesis or orogastric lavage, and repeated doses of activated charcoal every 4 to 6 hours for 3 days, because bromethalin undergoes enterohepatic recirculation. Supportive care includes intravenous fluids, anticonvulsants, muscle relaxants (methocarbamol up to 220 mg/kg/day IV to effect), frequent turning of the patient, and passive range-of-motion exercises. Supplemental oxygen and /or mechanical ventilation may be required in patients with coma and severe hypoventilation. Administer mannitol (0.5-1 g/kg) in conjunction with furosemide (1 mg/kg IV) if cerebral edema is suspected.

Caffeine*Introduction*

The majority of caffeine toxicities occur in dogs that ingest coffee beans. Caffeine causes phosphodiesterase inhibition, and can cause cardiac tachyarrhythmias, CNS stimulation (hyperexcitability and seizures), diuresis, gastric ulcers, vomiting, and diarrhea. Muscle tremors and seizures can occur, resulting in severe hyperthermia.

Treatment

Treatment of caffeine toxicity is largely symptomatic and supportive, as there is no known antidote. If clinical signs are not apparent and the patient is able to protect its airway, induce emesis. Alternatively, orogastric lavage can be performed, followed by administration of activated charcoal. Administer diazepam to control seizures. Administer beta-adrenergic blockers (e.g., esmolol, propranolol, atenolol) to control tachyarrhythmias. Give intravenous fluids to maintain hydration and correct hyperthermia. The patient should be walked frequently or have a urinary catheter placed to prevent reabsorption of the toxin from the urinary bladder.

Carbamates*Introduction*

Carbamate compounds are found in agricultural and home insecticide products. Examples of carbamates include carbofuran, aldicarb, propoxur, carbaryl, and methiocarb. The toxic dose of each compound varies. Carbamate compounds function by causing acetylcholinesterase inhibition. Toxic amounts cause CNS excitation, muscarinic acetylcholine overload, and SLUD (salivation, lacrimation, urination, and defecation). Miosis, vomiting,

and diarrhea result from muscarinic overload. Nicotinic overload produces muscle tremors. Toxicity can result in seizures, coma, and death.

Treatment

Treatment of carbamate intoxication includes maintaining an airway and, if necessary, artificial ventilation. Administer intravenous crystalloid fluids to control the patient's hydration, blood pressure, and temperature. Cooling measures may be warranted. Induce emesis if the substance was ingested within 60 minutes and the animal is asymptomatic. Give repeated doses of activated charcoal if the animal can swallow and protect its airway. Control seizures with diazepam (0.5 mg/kg IV). Bathe the patient thoroughly. Atropine (0.2 mg/kg IV) is useful in controlling some of the muscarinic signs associated with the toxicity. Pralidoxime hydrochloride (2-PAM) is not useful in cases of carbamate intoxication. Control muscle tremors with methocarbamol (up to 220 mg/kg IV) or guaifenesin.

Carbon tetrachloride

Introduction

In humans, ingestion or inhalation of 3-5 mL of carbon tetrachloride can be fatal. Clinical signs of carbon tetrachloride toxicity include vomiting and diarrhea, then progressive respiratory and central nervous system depression. Ventricular dysrhythmias and hepatorenal damage ensue. The prognosis is grave.

Treatment

Treatment of carbon tetrachloride inhalation includes procurement and maintenance of a patent airway with supplemental oxygen, and cardiovascular support. To treat ingestion, administer activated charcoal, and give intravenous fluids to maintain hydration and support renal function.

Chlorinated hydrocarbons

Introduction

Chlorinated hydrocarbons include DDT, methoxychlor, lindane, dieldrin, aldrin, chlordane, chlordecone, perthane, toxaphene, heptachlor, mirex, and endosulfan. The toxic dose of each compound varies. Chlorinated hydrocarbons exert their toxic effects by an unknown mechanism, and can be absorbed through the skin and the gastrointestinal tract. Clinical signs are similar to those observed in organophosphate toxicity: CNS excitation, seizures, SLUD, (salivation, lacrimation, urination, defecation), excessive bronchial secretions, vomiting, diarrhea, muscle tremors, and respiratory paralysis. Secondary toxicity from toxic metabolites can cause renal and hepatic failure. Chronic exposure may cause anorexia, vomiting, weight loss, tremors, seizures, and hepatic failure. The clinical course can be prolonged in small animal patients.

Treatment

Treatment of chlorinated hydrocarbon toxicity is largely supportive in nature, as there is no known antidote. Procure and maintain the patient's airway. Normalize the body temperature to prevent hyperthermia. If the substance was just ingested and the patient is not demonstrating any clinical signs, induce emesis. If the patient is symptomatic, perform orogastric lavage followed by activated charcoal administration. Bathe the patient thoroughly in cases of topical exposure. Administer intravenous crystalloid fluids to maintain hydration. These compounds do not appear to be amenable to fluid diuresis.

Chlorophenoxy herbicides

Introduction:

Chlorophenoxy derivatives are found in 2,4-D, 2,4,5-T, MCPA, MCPB, and Silvex. The LD₅₀ of 2,4-D is 100 mg/kg; however, the toxic dose appears to be much lower in small

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animal patients. Chlorphenoxy derivatives exert their toxic effects by an unknown mechanism, and cause clinical signs of gastroenteritis and muscle rigidity.

Treatment

Treatment of chlorphenoxy derivative toxicity is largely supportive in nature, as there is no known antidote. Secure the patient's airway and administer supplemental oxygen, as necessary. Control CNS excitation with diazepam (0.5 mg/kg IV). Intravenous crystalloid fluid diuresis and urinary alkalization can promote elimination. Administer gastroprotectant and antiemetic drugs, as needed.

Chocolate

Introduction

The toxic effects of chocolate are related to theobromine. Various types of chocolate have different concentrations of theobromine and thus can cause clinical signs of toxicity with ingestion of varying amounts of chocolate, depending on the type. The toxic dose of theobromine is 100-150 mg/kg in dogs. Milk chocolate contains 44 mg/oz (154 mg/100 g) of chocolate, and has a low toxic potential. Semisweet chocolate contains 150 mg/oz (528 mg/100 g), and baking chocolate contains 390 mg/oz (1365 mg/100 g). Semisweet and baking chocolate, being the most concentrated, have a moderate to severe toxic potential, even in large dogs.

Clinical signs of theobromine intoxication are associated with phosphodiesterase inhibition and include CNS stimulation (tremors, anxiety, seizures), myocardial stimulation (tachycardia and tachyarrhythmias), diuresis, and (at very high doses) gastrointestinal ulceration. With treatment, the condition of most dogs returns to normal within 12 to 24 hours ($t_{1/2}$ = 17.5 hours in dogs). Potential side effects include gastroenteritis and pancreatitis due to the fat content of the chocolate.

Treatment

Treatment of chocolate toxicity includes obtaining and maintaining a protected airway (if necessary), intravenous fluid diuresis, induction of emesis or orogastric lavage followed by administration of repeated doses of activated charcoal, and placement of a urinary catheter to prevent reabsorption of the toxin from the urinary bladder.

Cholecalciferol

Introduction

Cholecalciferol rodenticide ingestion can lead to increased intestinal and renal reabsorption of calcium, causing an increase in serum calcium and dystrophic mineralization of the kidneys and liver at 2-3 mg/kg. Clinical signs include lethargy, anorexia, vomiting, constipation, and renal pain within 2 to 3 days of ingestion. Seizures, muscle twitching, and central nervous system depression may be observed at very high doses. As renal failure progresses, polyuria, polydipsia, vomiting/hematemesis, uremic oral ulcers, and melena may be observed.

Treatment

If the compound was ingested recently (within 2 to 4 hours) induce emesis or perform orogastric lavage, followed by administration of activated charcoal. Check the patient's serum calcium once daily for three days following ingestion. If clinical signs of toxicity or hypercalcemia are present, decrease serum calcium with loop diuretics (furosemide, 2-5 mg/kg PO or IV q12h) and glucocorticosteroids (prednisone or prednisolone, 2-3 mg/kg PO bid) to promote renal calcium excretion. In severe cases, salmon calcitonin (4-6 IU/kg SC q2-12h in dogs) or bisphosphonate compounds may be required. Correct acid-base abnormalities with intravenous crystalloid fluid diuresis and sodium bicarbonate, if necessary. (See section on Hypercalcemia.)

Coal, tar-based: see hydrocarbons, aromatic

Coumarins: see vitamin K antagonist rodenticides

Cresol: see hydrocarbons, aromatic

Deicers: see ethylene glycol and alcohols

Denture cleaners

Introduction

Denture cleaners contain sodium perborate as the active compound. Sodium perborate can cause severe direct irritation of the mucous membranes and may also act as a CNS depressant. Clinical signs are similar to those seen if bleach or boric acid compound is ingested, namely vomiting, diarrhea, CNS excitation then depression, and renal failure.

Treatment

Treatment for ingestion of denture cleaner includes gastric decontamination along with induction of emesis or orogastric lavage and administration of a cathartic to hasten elimination. Activated charcoal is not useful for treatment of ingestion of this toxin. Administer intravenous fluid therapy to maintain renal perfusion. Administer gastroprotectant and antiemetic drugs, as necessary.

Deodorants

Introduction

Deodorants are usually composed of aluminum chloride and aluminum chlorohydrate. Both have a moderate potential for toxicity. Ingestion of deodorant compounds can cause oral irritation or necrosis, gastroenteritis, and nephrosis.

Treatment:

Treatment of deodorant ingestion includes orogastric lavage, and administration of antiemetic and gastroprotectant drugs.

Detergents, anionic

Introduction

Anionic detergents include sulfonated or phosphorylated forms of benzene. Dishwashing liquid is an example of an anionic detergent that can be toxic at doses of 1 – 5 g/kg. Anionic detergents cause significant mucosal damage and edema, gastrointestinal irritation, CNS depression, seizures, and possible hemolysis. Ocular exposure can cause corneal ulcers and edema.

Treatment

Treatment of anionic detergent exposure is largely symptomatic, as there is no known antidote. To treat topical toxicity, flush the patient's eyes and skin with warmed tap water or 0.9% saline solution for a minimum of 30 minutes, taking care to avoid hypothermia. To treat ingestion, feed the patient milk and large amounts of water to dilute the toxin. Do NOT induce emesis, because of the risk of worsening esophageal irritation. To dilute the toxin, perform orogastric lavage, followed by administration of activated charcoal. Closely monitor the patient's respiratory status, because oropharyngeal edema can be severe. If necessary, perform endotracheal intubation in cases of airway obstruction. Monitor the patient for signs of intravascular hemolysis. Administer intravenous crystalloid fluids to maintain hydration until the patient is able to tolerate oral fluids.

Detergents, cationic, and disinfectants

Introduction

Cationic detergents and disinfectants include quaternary ammonia compounds, isopropyl alcohol, and isopropanol. Quaternary ammonia compounds have a serious toxic potential

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and cause severe irritation and corrosion of the mucous membranes and skin. Some compounds also can cause clinical signs similar to those observed with anticholinesterase compounds, including muscle tremors, seizures, paralysis, and coma. Methemoglobinemia can occur.

Treatment

Treatment of cationic detergent exposure includes careful bathing and ocular rinsing of the patient for a minimum of 30 minutes, taking care to avoid hypotension. Secure the patient's airway and monitor the patient's respiratory status. Administer supplemental oxygen, if necessary. Place an intravenous catheter and administer intravenous crystalloid fluids to maintain hydration. Do NOT induce emesis, because of the risk of causing further esophageal irritation. Give milk or large amounts of water orally, as tolerated by the patient, to dilute the toxin.

Detergents, nonionic*Introduction*

Nonionic detergents include alkyl and aryl polyether sulfates, alcohols, and sulfonates; alkyl phenol; polyethylene glycol; and phenol compounds. Phenols are particularly toxic in cats and puppies. Clinical signs of exposure include severe gastroenteritis and topical irritation. Some compounds can be metabolized to glycolic and oxalic acid, causing renal damage similar to that observed with ethylene glycol toxicity.

Treatment

Topical and ocular exposure should be treated with careful bathing or ocular irrigation for at least 30 minutes. Administer activated charcoal to prevent absorption of the compound. As tolerated, give dilute milk or straight tap water orally to dilute the compound. Administer antiemetic and gastroprotectant drugs to control vomiting and decrease gastrointestinal irritation. Administer intravenous crystalloid fluids to maintain hydration and decrease the potential for renal tubular damage. Monitor the patient's acid-base and electrolyte status and correct any abnormalities with appropriate intravenous fluid therapy.

Dichlone*Introduction*

Diclone (Phigone) is a dipirydil compound that is a CNS depressant. The LD₅₀ in rats is 25-50 mg/kg. Dichlone reacts with thiol enzymes to cause methemoglobinemia and hepatorenal damage.

Treatment

To treat dichlone ingestion, induce emesis or perform orogastric lavage, followed by administration of activated charcoal and a cathartic. Procure and maintain a patent airway. Perform intravenous fluid diuresis to maintain renal perfusion. *N*-acetylcysteine may be useful in the treatment of methemoglobinemia.

Diethyltoluamide (DEET)*Introduction*

Diethyltoluamide (DEET) is the active ingredient in many insect repellants (e.g., Off, Cutters, Hartz Blockade). The mechanism of action of DEET is not fully understood, but it acts as a lipophilic neurotoxin within 5 to 10 minutes of exposure. Cats appear to be particularly sensitive to DEET. A lethal dermal dose is 1.8 g/kg; if ingested, the lethal dose is much less. The toxic dose of dermal exposure in dogs is 7 g/kg. Clinical signs of toxicity include aimless gazing, hypersalivation, chewing motions, and muscle tremors that progress to seizures. Recumbency and death can occur within 30 minutes of exposure at high doses.

Treatment

Treatment of DEET toxicity is largely supportive, as there are no known antidotes. Procure and maintain a patent airway and perform mechanical ventilation, if necessary. Place an intravenous catheter and administer intravenous crystalloid fluids to control hydration and treat hypotension, as necessary. Treat seizures with diazepam (0.5 mg/kg IV) or phenobarbital. Because of the rapid onset of clinical signs, induction of emesis is contraindicated. Perform orogastric lavage if the compound was ingested within the last 2 hours. Administer multiple repeated doses of activated charcoal. Cooling measures should be implemented to control hyperthermia. If dermal exposure has occurred, bathe the patient thoroughly to avoid further exposure and absorption.

Diquat

Introduction

Diquat is a dipyrindyl compound that is the active ingredient in some herbicide compounds. The LD₅₀ of diquat is 25–50 mg/kg. Like paraquat, diquat induces its toxic effects by causing the production of oxygen-derived free radical species. Clinical signs of diquat intoxication include anorexia, vomiting, diarrhea, and acute renal failure. Massive dehydration and electrolyte imbalances can occur as a result of fluid loss into the gastrointestinal tract.

Treatment

Treatment of diquat intoxication is similar to that for paraquat ingestion. If the animal had ingested diquat within 1 hour of presentation, induce emesis. In clinical cases, orogastric lavage may be required. Both emesis and orogastric lavage should be followed by administration of kaolin or bentonite as an adsorbent, rather than activated charcoal. Place an intravenous catheter and administer crystalloid fluids to restore volume status and maintain renal perfusion. Monitor urine output. If oliguria or anuria occurs, treatment with mannitol, furosemide, and dopamine may be considered.

Ecstasy

Introduction

Ecstasy (3,4-methylenedioxymethylamphetamine; MDMA) is a recreational drug used by humans. Ecstasy causes release of serotonin. Clinical signs of intoxication are related to the serotonin syndrome (excitation, hyperthermia, tremors, and hypertension), and seizures may be observed. A urine drug screening test can be used to detect the presence of MDMA.

Treatment

Treatment of ecstasy intoxication is largely supportive, as there is no known antidote. Administer intravenous fluids to maintain hydration, correct acid-base status, and treat hyperthermia. Serotonin antagonist drugs (cyproheptadine) can be dissolved and administered per rectum to alleviate clinical signs. Intravenous propranolol has additional anti-serotonin effects. Administer diazepam (0.5–2 mg/kg IV) to control seizures. If cerebral edema is suspected, administer mannitol, followed by furosemide.

Ethylene glycol

Introduction

Ethylene glycol is most commonly found in antifreeze solutions but is also in some paints, photography developer solutions, and windshield wiper fluid. Ethylene glycol in itself is only minimally toxic. However, when it is metabolized to glycolate, glyoxal, glyoxylate, and oxalate, the metabolites cause an increased anion gap metabolic acidosis and precipitation of calcium oxalate crystals in the renal tubules, renal failure, and (ultimately) death.

The toxic dose in dogs is 6.6 mL/kg, and in cats is 1.5 mL/kg. The toxin is absorbed quite readily from the gastrointestinal tract and can be detected in the patient's serum within an hour of ingestion. Colorimetric tests that can be performed in most veterinary hospitals can detect larger quantities of ethylene glycol in the patient's serum. In a dog with clinical

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signs of ethylene glycol intoxication and renal impairment or failure, a negative test for the presence of calcium oxalate crystalluria means that there is no more ethylene glycol in the patient's serum because it has all been metabolized. Cats are very sensitive to the toxic effects of ethylene glycol. In many cases, cat may have ingested a toxic dose, but because the sensitivity of the assay is low, test results will be negative. Lack of treatment can result in death.

There are three phases of ethylene glycol intoxication. In the first 1 to 12 hours after ingestion (stage I), the patient may appear lethargic, disoriented, and ataxic. In stage II (12 to 24 hours following ingestion), the patient improves and appears clinically normal. In stage III (24 to 72 hours following ingestion), the patient demonstrates clinical signs of renal failure (polyuria and polydipsia) that progress to uremic renal failure (vomiting, lethargy, oral ulceration). Finally, seizures, coma, and death occur.

Treatment

Begin treatment of known ethylene glycol ingestion immediately. Induce emesis or perform orogastric lavage and administer repeated doses of activated charcoal. Place an intravenous catheter and perform crystalloid fluid diuresis with a known antidote. The treatment of choice for dogs is administration of 4-methylpyrazole (4-MP), which directly inhibits alcohol dehydrogenase, thus preventing the conversion of ethylene glycol to its toxic metabolites. The dose for dogs is 20 mg/kg initially, followed by 15 mg/kg at 12 and 24 hours and 5 mg/kg at 36 hours. 4-MP has been used experimentally at 6.25 times the recommended dose for dogs. In cats, treatment with 4-MP is effective if it is administered within the first 3 hours of ingestion.

Cats will demonstrate signs of sedation and hypothermia with this treatment. If 4-MP is not available, administer ethanol (600 mg/kg IV loading dose, followed by 100 mg/kg/hour), or as a 20% solution (for dogs, 5.5 mL/kg IV q4h for five treatments, then q 6h for five more treatments; for cats, 5 mL/kg q8h for four treatments). Grain alcohol (190 proof) contains approximately 715 mg/mL of ethanol. Antiemetics and gastroprotective agents should be considered. Urinary alkalization and peritoneal dialysis may enhance the elimination of ethylene glycol and its metabolites.

Fertilizers

Introduction

Many fertilizers are on the market, and may be composed of urea or ammonium salts, phosphates, nitrates, potash, and metal salts. Fertilizers have a moderate toxic potential, depending on the type and amount ingested. Clinical signs of fertilizer ingestion include vomiting, diarrhea, metabolic acidosis, and diuresis. Nitrates or nitrites can cause formation of methemoglobin and chocolate-brown blood. Electrolyte disturbances include hyperkalemia, hyperphosphatemia, hyperammonemia, and hyperosmolality.

Treatment

Treatment of fertilizer ingestion includes cardiovascular support, and administration of milk or a mixture of egg whites and water, followed by induction of emesis or orogastric lavage. Correct electrolyte abnormalities as they occur (see section on Hyperkalemia). Administer antiemetic and gastroprotectant drugs, as necessary. Administer intravenous fluids to control hydration and maintain blood pressure. *N*-acetylcysteine may be useful if methemoglobinemia is present.

Fipronil

Introduction

Fipronil is the active ingredient in Frontline, a flea control product. Fipronil exerts its effects by GABA antagonism and can cause CNS excitation.

Treatment

Treatment of fipronil toxicity includes treatment of CNS excitation, treatment of hyperthermia by cooling measures, and administration of activated charcoal.

Fire extinguisher (liquid)*Introduction*

Fire extinguisher fluid contains chlorobromomethane or methyl bromide, both of which have a serious toxic potential. Dermal or ocular irritation can occur. If ingested, the compounds can be converted to methanol, and cause high anion gap metabolic acidosis, CNS excitation and depression, aspiration pneumonitis, and hepatorenal damage.

Treatment

To treat ocular or dermal exposure to fire extinguisher fluids, flush the eyes or skin with warmed tap water or 0.9% saline solution for a minimum of 30 minutes. Do NOT induce emesis or perform orogastric lavage to treat ingestion, because of the risk of causing severe aspiration pneumonitis. Gastroprotectant and antiemetic drugs may be used, if indicated. Administer intravenous fluids to maintain hydration and renal perfusion. Supplemental oxygen or mechanical ventilation may be required in severe cases of aspiration pneumonitis.

Fireplace colors*Introduction*

Fireplace colors contain salts of heavy metals—namely, copper, rubidium, cesium, lead, arsenic, antimony, barium, selenium, and zinc, all of which have moderate toxic potential, depending on the amount ingested and the size of the patient. Clinical signs are largely associated with gastrointestinal irritation (vomiting, diarrhea, anorexia). Zinc toxicity can cause intravascular hemolysis and hepatorenal damage.

Treatment

To treat ingestion of fireplace colors, administer cathartics and activated charcoal and gastroprotectant and antiemetic drugs. Place an intravenous catheter for intravenous crystalloid fluid administration to maintain hydration and renal perfusion. Specific chelating agents may be useful in hastening elimination of the heavy metals.

Fireworks*Introduction*

Fireworks contain oxidizing agents (nitrates and chlorates) and metals (mercury, copper, strontium, barium, and phosphorus). Ingestion of fireworks can cause hemorrhagic gastroenteritis and methemoglobinemia.

Treatment

To treat firework ingestion, induce emesis or perform orogastric lavage and administer activated charcoal. Administer specific chelating drugs if the amount and type of metal are known, and administer gastroprotectant and antiemetic drugs. If methemoglobinemia occurs, administer *N*-acetylcysteine; a blood transfusion may be necessary.

Fuels*Introduction*

Fuels such as barbecue lighter fluid, gasoline, kerosene, and oils (mineral, fuel, lubricating) are petroleum distillate products that have a low toxic potential if ingested but can cause severe aspiration pneumonitis if as little as 1 mL is inhaled into the tracheobronchial tree. CNS depression, mucosal damage, hepatorenal insufficiency, seizures, and corneal irritation can occur.

Treatment

If fuels are ingested, administer gastroprotectant and antiemetic drugs. Do NOT induce emesis or perform orogastric lavage, because of the risk of aspiration pneumonia. To treat topical exposure, rinse the skin and eyes copiously with warm tap water or

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0.9% saline solution. Administer antiemetic and gastroprotectant drugs, as necessary. Administer intravenous fluids to maintain hydration and treat acid-base and electrolyte abnormalities.

Furniture polish: see fuels

Gasolines: see Fuels

Glue, children's

Introduction

Children's glue contains polyvinyl acetate, which has a very low toxic potential. If inhaled, the compound can cause pneumonitis.

Treatment

Treatment of polyvinyl acetate should be performed as clinical signs of pneumonitis (increased respiratory effort, cough, lethargy, respiratory distress) occur.

Glue, Superglue

Introduction

Superglue contains methyl-2-cyanoacrylate, a compound that can cause severe dermal irritation on contact.

Treatment:

Do NOT induce emesis. Do NOT bathe the animal, and do NOT apply other compounds (acetone, turpentine) in an attempt to remove the glue from the skin. The fur can be shaved, using care to avoid damaging the underlying skin. The affected area should be allowed to exfoliate naturally.

Glyphosate

Introduction

Glyphosate is a herbicide found in Roundup and Kleenup. If applied properly, the product has a very low toxic potential. Clinical signs of toxicity include dermal and gastric irritation, including dermal erythema, anorexia, and vomiting. CNS depression can occur.

Treatment

Treatment includes thorough bathing in cases of dermal exposure, and induction of emesis or orogastric lavage followed by administration of activated charcoal. Administer antiemetic and gastroprotectant drugs as necessary. Administer intravenous crystalloid fluids to prevent dehydration secondary to vomiting.

Grapes and raisins

Introduction

Even small amounts of grapes and raisins can be toxic to dogs. The mechanism of toxicity remains unknown. Clinical signs occur within 24 hours of ingestion of raisins or grapes, and include vomiting, anorexia, lethargy, and diarrhea (often with visible raisins or grapes in the fecal matter). Within 48 hours, dogs demonstrate signs of acute renal failure (polyuria, polydipsia, vomiting) that can progress to anuria.

Treatment

To treat known ingestion of raisins or grapes, induce emesis or perform orogastric lavage, followed by repeated doses of activated charcoal. If clinical signs of vomiting and diarrhea are present, administer intravenous fluids and monitor urine output. Aggressive intravenous fluid therapy, in conjunction with maintenance of renal perfusion, is necessary. In cases of anuric renal failure, dopamine, furosemide, and mannitol can be useful in increasing urine output. Peritoneal or hemodialysis may be necessary in cases of severe oliguric or anuric renal failure. Calcium channel blockers such as amlodipine and diltiazem can be used to treat systemic hypertension. Supportive care includes treatment of hyperkalemia,

and administration of gastroprotectant and antiemetic drugs and (if the animal is eating) phosphate binders.

Hashish: see marijuana

Hexachlorophene: see detergents, nonionic

Hydrocarbons, aromatic

Introduction

Aromatic hydrocarbons include phenols, cresols, toluene, and naphthalene. All have a moderate toxic potential if ingested. Toxicities associated with ingestion of aromatic hydrocarbons include CNS depression, hepatorenal damage, muscle tremors, pneumonia, methemoglobinemia, and intravascular hemolysis.

Treatment

If an aromatic hydrocarbon is ingested, do NOT induce emesis, because of the risk of aspiration pneumonia. A dilute milk solution or water can be administered to dilute the compound. Perform orogastric lavage. Carefully monitor the patient's respiratory and cardiovascular status. Administer supplemental oxygen if aspiration pneumonia is present. To treat topical exposure, thoroughly rinse the eyes and skin with copious amounts of warm tap water or 0.9% saline solution.

Ibuprofen: see nonsteroidal antiinflammatory drugs

Imidacloprid

Introduction

Imidacloprid is the compound used in the flea product Advantage. Clinical signs of toxicity are related to nicotinic cholinergic stimulation, causing neuromuscular excitation followed by collapse. The compound may induce respiratory paralysis.

Treatment

To treat imidacloprid toxicity, procure and maintain a patent airway with supplemental oxygen administration. Control CNS excitation with diazepam, phenobarbital, or propofol. Administer enemas to hasten gastrointestinal elimination, and administer activated charcoal. Bathe the animal thoroughly to prevent further dermal absorption. Closely monitor the patient's oxygenation and ventilation status. If severe hypoventilation or respiratory paralysis occurs, initiate mechanical ventilation.

Iron and iron salts

Introduction

Iron and iron salts can cause severe gastroenteritis, myocardial toxicity, and hepatic damage if high enough doses are ingested. Lawn fertilizers are a common source of iron salts.

Treatment:

Treatment of ingestion of iron and iron salts includes cardiovascular support in the form of intravenous fluids and antiarrhythmic drugs, as needed. Induce emesis or perform orogastric lavage for gastric decontamination. A cathartic can be administered to promote elimination from the gastrointestinal tract. Antiemetic and gastroprotectant drugs should be administered to prevent nausea and vomiting. In some cases, radiographs can aid in making a diagnosis of whether the compound was actually ingested. Iron toxicity can be treated with the chelating agent deferoxamine.

Ivermectin

Introduction

Ivermectin is a GABA agonist that is used in commercial heartworm prevention and antihelminthic compounds and can be toxic in predisposed breeds, including Collies, Collie

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crosses, Old English Sheepdogs, and some Terriers. Clinical signs of ivermectin toxicity include vomiting, ataxia, hypersalivation, agitation, tremors, hyperactivity, hyperthermia, hypoventilation, coma, seizures, signs of circulatory shock, bradycardia, and death. Clinical signs often occur within 2 to 24 hours after ingestion or iatrogenic overdose. Blood ivermectin levels can be measured, but diagnosis is often made based on clinical signs and knowledge of exposure in predisposed breeds. There is no known antidote. The clinical course can be prolonged for weeks to months before recovery occurs.

Treatment

To treat known exposure, induce emesis or perform orogastric lavage if the substance was ingested within 1 hour of presentation and the patient is not symptomatic. Administer activated charcoal. Control seizures with phenobarbital, pentobarbital, or propofol administered as intermittent boluses or as a constant rate infusion. Diazepam, which potentially can *worsen* central nervous stimulation, is contraindicated. Administer intravenous fluids to maintain perfusion and hydration, and treat hyperthermia. Supportive care may be necessary, including supplemental oxygen (or mechanical ventilation, if necessary), frequent turning of the patient and passive range-of-motion exercises, placement of a urinary catheter to maintain patient cleanliness and monitor urine output, lubrication of the eyes, and parenteral nutrition (see section on Rule of Twenty). Specific antidotes used to treat ivermectin toxicity include physostigmine and picrotoxin. Physostigmine therapy was beneficial in some patients for a short period; picrotoxin caused severe violent seizures and therefore should be avoided.

Kerosene: see fuels

d-Limonene, linalool

Introduction

d-Limonene and linalool are components of citrus oil extracts used in some flea control products. The toxic dose is unknown, but cats appear to be very sensitive to exposure. Clinical signs of toxicity include hypersalivation, muscle tremors, ataxia, and hypothermia.

Treatment

Treatment of d-Limonene and linalool exposure includes treatment of hypothermia, administration of activated charcoal to prevent further absorption, and careful, thorough bathing to prevent further dermal exposure.

Lead

Introduction

Lead is ubiquitous, and is found in some paints, car batteries, fishing equipment/sinkers, and plumbing materials. Lead can be toxic at doses of 3 mg/kg. If more than 10-25 mg/kg of lead is ingested, death can occur. Lead causes toxicity by inhibiting sulfur-containing enzymes, leading to increased RBC fragility, and CNS damage. Clinical signs of hyperexcitability, dementia, vocalization, seizures, and lower motor neuron polyneuropathy can occur. Affected animals may appear blind, or vomiting, anorexia, and constipation or diarrhea may occur. If lead toxicity is suspected, blood and urine lead levels can be measured.

Treatment

Treatment of lead toxicity is supportive and is directed at treatment of clinical signs. Control seizures with diazepam or phenobarbital. If cerebral edema is present, administer mannitol (0.5-1.0 g/kg IV), followed by furosemide (1 mg/kg IV 20 minutes after mannitol). Sodium or magnesium sulfate should be administered as a cathartic. Initiate chelation therapy with dimercaprol, penicillamine, or calcium EDTA. If a lead object is identified in the gastrointestinal tract on radiographs, remove the object using endoscopy or exploratory laparotomy.

Loperamide*Introduction*

Loperamide is an opioid derivative that is used to treat diarrhea. Clinical signs of loperamide intoxication include constipation, ataxia, nausea, and sedation.

Treatment

Induce emesis or perform orogastric lavage, followed by administration of activated charcoal and a cathartic. Naloxone may be beneficial in the temporary reversal of ataxia and sedation.

Macadamia nuts*Introduction*

Ingestion of macadamia nuts can cause clinical signs of vomiting, ataxia, and ascending paralysis in dogs. The toxic principle in macadamia nuts is unknown.

Treatment

There is no known antidote. Treatment consists of supportive care, including administration of intravenous fluids and antiemetics and placement of a urinary catheter for patient cleanliness. Clinical signs resolve in most cases within 72 hours.

Marijuana (*Cannabis sativa*)*Introduction*

Marijuana is a hallucinogen that can cause CNS depression, ataxia, mydriasis, increased sensitivity to motion or sound, salivation, and tremors. Along with these findings, a classic clinical sign is the sudden onset of dribbling urine. Urine can be tested with drug test kits for tetrahydrocannabinoid (THC), the toxic compound in marijuana.

Treatment

There is no known antidote for marijuana toxicity; therefore, treatment is largely symptomatic. place an intravenous catheter and administer intravenous fluids to support hydration. Administer atropine if severe bradycardia exists. Induction of emesis can be attempted but because of the antiemetic effects of THC, is usually unsuccessful. Orogastric lavage can be performed, followed by repeated doses of activated charcoal. Clinical signs usually resolve within 12 to 16 hours.

Matches*Introduction*

“Strike Anywhere” matches, safety matches, and the striking surface of matchbook covers contain iron phosphorus or potassium chlorate. Both compounds have a low toxic potential but can cause clinical signs of gastroenteritis and methemoglobinemia if large quantities are ingested.

Treatment

Treatment of match and matchbook ingestion includes gastric decontamination with induction of emesis or orogastric lavage and administration of activated charcoal and a cathartic. If methemoglobinemia occurs, administer *N*-acetylcysteine, intravenous fluids, and supplemental oxygen.

Metaldehyde*Introduction*

Metaldehyde is the active ingredient in most brands of snail bait. The exact mechanism of toxicity is unknown but may involve inhibition of GABA channels. Clinical signs associated with metaldehyde toxicity include severe muscle tremors, CNS excitation, and

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hyperthermia, that occurs within 15 – 30 minutes of ingestion. Diarrhea and convulsions can develop. If hyperthermia is severe, renal failure secondary to myoglobinuria and disseminated intravascular coagulation can result. Delayed hepatic failure has been described days after initial recovery. If metaldehyde toxicosis is suspected, analysis of urine, serum, and stomach contents is warranted.

Treatment

To treat metaldehyde toxicity, procure and maintain a patent airway and control CNS excitation and muscle tremors. If an animal has just ingested the metaldehyde and is not symptomatic, induce emesis. If clinical signs are present, perform orogastric lavage. Both emesis and orogastric lavage should be followed by administration of one dose of activated charcoal. Administer intravenous fluids to control hyperthermia, prevent dehydration, and correct acid-base and electrolyte abnormalities. Methocarbamol is the treatment of choice to control muscle tremors. Diazepam can be used to control seizures if they occur.

Methiocarb: see carbamates

Mineral Spirits: see fuels

Mothballs: see naphthalene

Mushrooms

Introduction

Mushroom ingestion most commonly causes activation of the autonomic nervous system, resulting in tremors, agitation, restlessness, hyperexcitability, and seizures. In some cases SLUD (salivation, lacrimation, urination, and defecation) is seen. Some mushrooms (*Amanita* spp.) also can cause hepatocellular toxicity. Clinical signs include vomiting, anorexia, lethargy, and progressive icterus.

Treatment

Treatment of mushroom toxicity is largely supportive. If the mushroom was ingested within the last 2 hours, induce emesis or perform orogastric lavage and then administer activated charcoal. Symptomatic treatment includes intravenous fluids to promote diuresis and treat hyperthermia and skeletal muscle relaxants to control tremors and seizures (methocarbamol, diazepam). If *Amanita* ingestion is suspected, administer hepatoprotectant agents including milk thistle.

Mycotoxins (tremorigenic mycotoxins)

Introduction

Mycotoxins from *Penicillium* spp. are found in moldy foods, cream cheese, and nuts. Clinical signs of intoxication include tremors, agitation, hyperesthesia, and seizures. If tremorigenic mycotoxin toxicity is suspected, a sample of the patient's serum and gastric contents or vomitus can be submitted to the Michigan State University Veterinary Toxicology Laboratory for tremorigen assay.

Treatment

There is no known antidote. Perform orogastric lavage, followed by administration of activated charcoal. Control tremors and seizures with methocarbamol, diazepam, phenobarbital, or pentobarbital. Administer intravenous fluids to control hyperthermia and maintain hydration. In cases in which cerebral edema is suspected secondary to severe refractory seizures, administer intravenous mannitol and furosemide.

Naphthalene

Introduction

Naphthalene is the active ingredient in mothballs and has a high toxic potential. Clinical signs associated with naphthalene toxicity include vomiting, methemoglobinemia, CNS

stimulation, seizures, and hepatic toxicity. A complete blood count often reveals Heinz bodies and anemia.

Treatment

DO NOT induce emesis if naphthalene ingestion is suspected. If the ingestion was within 1 hour of presentation, perform orogastric lavage. Control seizures with diazepam or phenobarbital. Administer intravenous fluids to control hyperthermia and maintain hydration. *N*-acetylcysteine can play a role in the treatment of methemoglobinemia. A packed RBC transfusion may be necessary if anemia is severe. Observe the patient for clinical signs associated with hepatitis.

Nicotine

Introduction

Nicotine toxicity occurs in animals as the result of ingestion of cigarettes, nicotine-containing gum, and some insecticides. Nicotine stimulates autonomic ganglia at low doses, and blocks autonomic ganglia and the neuromuscular junction at high doses. Absorption after ingestion is rapid. Clinical signs include hyperexcitability and SLUD (salivation, lacrimation, urination, and defecation). Muscle tremors, respiratory muscle fatigue or hypoventilation, tachyarrhythmias, seizures, coma, and death can occur.

Treatment

If the patient presents within 1 hour of ingestion and has no clinical signs, induce emesis, followed by administration of repeated doses of activated charcoal. In patients with clinical signs of toxicity, perform orogastric lavage. Administer intravenous fluids to maintain hydration and promote diuresis, and treat hyperthermia. Administer atropine to treat cholinergic symptoms. Urinary acidification can promote nicotine excretion.

Nonsteroidal ant-inflammatory drugs

Introduction

Nonsteroidal antiinflammatory drugs (NSAIDs) include ibuprofen, ketoprofen, carprofen, diclofenac, naproxen, celecoxib, valdecoxib, rofecoxib, and deracoxib. NSAIDs cause inhibition of prostaglandin synthesis, leading to gastrointestinal ulceration, renal failure and hepatotoxicity. Ibuprofen toxicity has been associated with seizures in dogs, cats, and ferrets. The toxic dose varies with the specific compound ingested.

Treatment

To treat NSAID toxicity, induce emesis or perform orogastric lavage, followed by administration of multiple repeated doses of activated charcoal. Place an intravenous catheter for crystalloid fluid diuresis to maintain renal perfusion. Administer the synthetic prostaglandin analogue misoprostol to help maintain gastric and renal perfusion. Control seizures, if present, with intravenous diazepam. Administer gastroprotectant and antiemetic drugs to control vomiting and gastrointestinal hemorrhage. Continue intravenous fluid diuresis for a minimum of 48 hours, with frequent monitoring of the patient's BUN and creatinine. When the BUN and creatinine levels are normal or have plateaued for 24 hours, slowly decrease fluid diuresis 25% per day until maintenance levels are restored.

Oils (lubricating, fuel, mineral): see fuels

Onions, garlic, and chives

Introduction

Onions, garlic, and chives contain sulfoxide compounds that can cause oxidative damage of RBCs, leading to Heinz body anemia, methemoglobinemia, and intravascular hemolysis. Clinical signs of toxicity include weakness, lethargy, tachypnea, tachycardia, and pale mucous membranes. Vomiting and diarrhea can occur. Intravascular hemolysis can cause

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hemoglobinuria and pigment damage of the renal tubular epithelium. Heinz bodies may be observed on cytologic evaluation of the peripheral blood smear.

Treatment

Treatment of onion, chive, and garlic toxicity includes administration of intravenous fluid diuresis, and induction of emesis or orogastric lavage, followed by administration of activated charcoal and a cathartic. In cases of severe anemia, packed RBC transfusion or administration of a hemoglobin-based oxygen carrier should be considered.

Opiates*Introduction*

Opiate drugs include heroin, morphine, oxymorphone, fentanyl, meperidine, and codeine. Opiate compounds bind to specific opioid receptors throughout the body and produce clinical signs of miosis or mydriasis (cats), and CNS excitation, followed by ataxia and CNS depression, leading to stupor and coma. Hypoventilation, bradycardia, hypoxia, and cyanosis can occur.

Treatment

To treat known overdose or ingestion of an opiate compound, induce emesis (in asymptomatic animals) or perform orogastric lavage, followed by administration of activated charcoal. Administer intravenous fluids and supplemental oxygen to support the cardiovascular and respiratory systems. Mechanical ventilation may be necessary until hypoventilation resolves. Administer repeated doses of naloxone as a specific antidote to reverse clinical signs of narcosis and hypoventilation. If seizures are present (meperidine toxicity), administer diazepam.

Oral contraceptives**Organophosphates***Introduction*

Organophosphate compounds traditionally are used in flea control products and insecticides. Common examples of organophosphates include chlorpyrifos, coumaphos, diazinon, dichlorvos, and malathion. The toxic dose varies, depending on the particular compound and individual animal sensitivity. Organophosphate toxicity causes acetylcholinesterase inhibition, resulting in clinical signs of CNS stimulation, including tremors and seizures. Muscarinic acetylcholine overload causes the classic SLUD signs of salivation, lacrimation, urination, and defecation. Miosis, excessive bronchial secretions, muscle tremors, and respiratory paralysis can occur. An intermediate syndrome of generalized weakness, hypoventilation, and eventual paralysis with ventral cervical ventroflexion that may require mechanical ventilation has been described. If organophosphate toxicity is suspected, whole-blood acetylcholinesterase activity can be measured and will be low.

Treatment

Treatment of toxicity includes careful and thorough bathing in cases of dermal exposure and, if the substance was ingested, gastric decontamination with induction of emesis or orogastric lavage, followed by administration of activated charcoal, and administration of the antidote pralidoxime hydrochloride (2-PAM). Atropine can help control the muscarinic clinical signs. Supportive care in the form of cooling measures, intravenous crystalloid fluids, and supplemental oxygen or mechanical ventilation may be required, depending on the severity of clinical signs.

Paint and varnish removers: see fuels**Paints and varnishes: see fuels****Paintballs***Introduction*

Ingestion of large amounts of paintballs can cause neurologic signs, electrolyte abnormalities, and occasionally death. Paintballs are gelatin capsules that contain multiple colors of

paint in a sorbitol or glycerol carrier. When large quantities of these osmotically active sugars are ingested, osmotic shifts of fluid cause a sudden onset of neurologic or gastrointestinal signs, including ataxia, seizures, and osmotic diarrhea caused by massive fluid shifts into the gastrointestinal tract. The loss of water in excess of solute can result in hypernatremia, a free water deficit, and increased serum osmolality.

Treatment

Following orogastric lavage, treatment of ingestion includes administering warm water enemas to help speed the movement of the paintballs through the gastrointestinal tract. Do NOT administer activated charcoal (usually in a propylene glycol carrier), because the compound's cathartic action will pull more fluid into the gastrointestinal tract. Baseline electrolytes should be obtained and then carefully monitored. If severe hypernatremia develops, administer hypotonic solutions such as 0.45% NaCl + 2.5% dextrose or 5% dextrose in water after calculating the patient's free water deficit. Because of the large volume of fluid loss, intravenous fluid rates may seem excessive but are necessary to normalize acid-base, electrolyte, and hydration status. In most cases, these patients can survive if the problem is recognized promptly and corrected with careful electrolyte monitoring, aggressive decontamination strategies, and intravenous fluid support.

Paracetamol: see acetaminophen

Paraquat

Introduction

Paraquat, a dipyridyl compound, is the active ingredient in some herbicides. The LD₅₀ of paraquat is 25-50 mg/kg. Paraquat initially causes CNS excitation. It also causes production of oxygen-derived free radical species in the lungs, that can lead to the development of acute respiratory distress syndrome. Initial clinical signs include vomiting, diarrhea, and seizures. Within 2 to 3 days, clinical signs associated with severe respiratory distress and acute respiratory distress syndrome (ARDS) can develop, leading to death. Chronic effects include pulmonary fibrosis, if the patient survives the initial toxicity period. The prognosis for paraquat toxicity is generally unfavorable.

Treatment

To treat Paraquat ingestion, remove the toxin from the gastrointestinal tract as rapidly as possible after ingestion. There are no known antidotes. If the compound was ingested within the past hour and the animal is able to protect its airway, induce emesis. Otherwise, perform orogastric lavage. Activated charcoal is not as effective as clay or bentonite adsorbents for removing this particular toxin. Early in the course of paraquat toxicity, oxygen therapy is contraindicated because of the risk of producing oxygen-derived free radical species. Later, oxygen therapy, including mechanical ventilation, is necessary if ARDS develops. Experimentally, free radical scavengers (*N*-acetyl cysteine, vitamin C, vitamin E, SAME) have been shown to be useful in preventing damage caused by oxygen-derived free radical species. Hemoperfusion may be useful in eliminating the toxin, if it is performed early in the course of toxicity.

Paraffin wax: see fuels

Pennies: see zinc and zinc oxide

Pennyroyal oil

Introduction

Pennyroyal oil is an herbal flea control compound that contains menthofuran as its toxic compound. Menthofuran is hepatotoxic and may cause gastrointestinal hemorrhage and coagulopathies.

Treatment

To treat toxicity, administer a cathartic and activated charcoal and antiemetic and gastro-protectant drugs, and thoroughly bathe the animal to prevent further dermal exposure.

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Petroleum distillates: see fuels

Phenobarbital: see barbiturates

Phenylcyclidine (angel dust)

Introduction

Phenylcyclidine (Angel Dust) is an illicit recreational drug that causes both CNS depression and excitation, decreased cardiac output, and hypotension.

Treatment

To treat phenylcyclidine toxicity, place an intravenous catheter, and administer intravenous fluids and antiarrhythmic drugs to maintain organ perfusion. Administer supplemental oxygen, and administer diazepam to control seizures. Urine alkalization can help eliminate the compound.

Phenylephrine

Introduction

Phenylephrine is an α -adrenergic agonist in many over-the-counter decongestant preparations. Clinical signs of intoxication include mydriasis, tachypnea, agitation, hyperactivity, and abnormal flybiting and staring behavior. Tachycardia, bradycardia, hypertension, hyperthermia, and seizures can occur.

Treatment:

To treat phenylephrine toxicity, place an intravenous catheter and give intravenous fluids to maintain hydration, promote diuresis, and treat hyperthermia. Administer prazosin or sodium nitroprusside to treat hypertension, antiarrhythmic drugs as necessary, and diazepam to control seizures.

Phenylpropanolamine

Introduction

Phenylpropanolamine has both α - and β -adrenergic agonist effects, and is used primarily in the treatment of urinary incontinence in dogs. The drug was taken off of the market for use in humans because of the risk of stroke. Clinical signs of phenylpropanolamine intoxication include hyperactivity, hyperthermia, mydriasis, tachyarrhythmias or bradycardia, hypertension, agitation, and seizures.

Treatment

To treat toxicity, administer prazosin or nitroprusside to control hypertension, a beta-blocker (esmolol, propranolol, atenolol) to control tachyarrhythmias, diazepam to control seizures, and intravenous fluids to maintain hydration and promote diuresis. Urine acidification may aid in facilitating excretion. If bradycardia occurs, do NOT use atropine.

Pseudoephedrine

Introduction

Pseudoephedrine is an α - and β -adrenergic agonist that is a component of many over-the-counter decongestants and is used in the manufacture of crystal methamphetamine. Clinical signs of toxicity include severe restlessness, tremors, mydriasis, agitation, hyperthermia, tachyarrhythmias or bradycardia, hypertension, and seizures.

Treatment

To treat toxicity, administer activated charcoal, intravenous fluids to promote diuresis and treat hyperthermia, chlorpromazine to combat α -adrenergic effects, a beta-blocker (propranolol, esmolol, atenolol) to treat β -adrenergic effects, and cyproheptadine (per rectum) to combat serotonergic effects.

Photographic developer solutions: see detergents, nonionic

Pine oil disinfectants: see detergents, nonionic, and alcohols

Piperazine

Introduction

Piperazine is a GABA agonist, and causes cervical and truncal ataxia, tremors, seizures, coma, and death.

Treatment

If ingestion was recent and if no clinical signs of toxicity are present, induce emesis or perform orogastric lavage, followed by administration of a cathartic and activated charcoal. There is no known antidote. Treatment includes supportive care in the form of intravenous fluids and administration of phenobarbital or methocarbamol to control seizures and tremors. Diazepam, a GABA agonist, is contraindicated, because it can potentially worsen clinical signs. Urine acidification may hasten elimination. Clinical signs can last from 3 to 5 days.

Pyrethrin and pyrethroids

Introduction

Pyrethrin and pyrethroid compounds are extracted from chrysanthemums, and include allethrin, decamethrin, tralomethrin, fenpropanthrin, palleshin, sumethrin, permethrin, tetramethrin, cyfluthrin, and resmethrin. The oral toxicity is fairly low; however, the compounds can be significantly harmful if inhaled or applied to the skin. Pyrethrin and pyrethroid compounds cause depolarization and blockade of nerve membrane potentials, causing clinical signs of tremors, seizures, respiratory distress, and paralysis. Contact dermatitis can occur. To distinguish between pyrethrin/pyrethroid toxicity and organophosphate toxicity, acetylcholinesterase levels should be obtained; they will be normal if pyrethrins are the cause of the animal's clinical signs.

Treatment

Treatment of toxicity is supportive, as there is no known antidote. Carefully bathe the animal in lukewarm water to prevent further oral and dermal exposure. Both hyperthermia and hypothermia can worsen clinical signs. Administer activated charcoal to decrease enterohepatic recirculation. Atropine may control clinical signs of excessive salivation. To control muscle tremors, administer methocarbamol to effect. Administer diazepam or phenobarbital to control seizures, as necessary.

Radiator fluids: see ethylene glycol

Raisins: see grapes and raisins

Rotenone

Introduction

Rotenone is used as a common garden and delousing insecticide. Fish and birds are very susceptible to rotenone toxicity. Rotenone inhibits mitochondrial electron transport. Clinical signs of tissue irritation and hypoglycemia can occur after topical or oral exposure. If the compound is inhaled, CNS depression and seizures can occur.

Treatment

To treat toxicity, perform orogastric lavage, followed by administration of a cathartic and activated charcoal. Bathe the animal carefully to prevent further dermal exposure and further ingestion. Administer diazepam or phenobarbital to control seizures. The prognosis generally is guarded.

Rubbing alcohol: see alcohols

Rust removers: see acids/corrosives

Salicylates: see aspirin

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Salt, thawing*Introduction*

Salt used for thawing ice commonly contains calcium chloride, a compound that has a moderate toxic potential. Calcium chloride produces strong local irritation and can cause gastroenteritis and gastrointestinal ulcers if ingested.

Treatment

Treatment of ingestion includes dilution with milk, water, or egg whites. Perform orogastric lavage, followed by administration of activated charcoal. Administer intravenous crystalloid fluids to maintain hydration. Administer antiemetic and gastroprotectant drugs to treat gastroenteritis and vomiting.

Shampoos, nonmedicated: see detergents, nonionic**Shampoos, selenium sulfide***Introduction*

Selenium sulfide shampoos (e.g., Selsun Blue) have a low toxic potential, and primarily cause gastroenteritis.

Treatment

Treatment of ingestion includes dilution with water, milk, or egg whites and administration of activated charcoal. Carefully and thoroughly rinse the skin and eyes to prevent further exposure. Administer antiemetic and gastroprotectant drugs in cases of severe gastroenteritis.

Shampoos, zinc-based (anti-dandruff)*Introduction*

Zinc-based (zinc pyridinethione) anti-dandruff shampoos have a serious toxic potential if ingested or if ocular exposure occurs. Gastrointestinal irritation, retinal detachment, progressive blindness, and exudative chorioretinitis can occur.

Treatment

Treatment of ingestion includes gastric decontamination. Induce emesis or perform orogastric lavage, followed by administration of a cathartic and activated charcoal.

To treat ocular exposure, thoroughly rinse the patient's eyes for a minimum of 30 minutes. Carefully monitor the animal for clinical signs of blindness. Implement intravenous fluid to maintain hydration and renal perfusion in cases of severe gastroenteritis.

Shoe polish: see aromatic hydrocarbons**Silver polish***Introduction*

Silver polish contains the alkali substance sodium carbonate and cyanide salts, and has a serious toxic potential. Ingestion results in rapid onset of vomiting and possibly cyanide toxicity.

Treatment

To treat ingestion, monitor and maintain the patient's respiration and cardiovascular status and administer intravenous crystalloid fluids. Induce emesis, followed by administration of activated charcoal. Administer sodium nitrite or sodium thiosulfate IV for cyanide toxicity.

Soaps (bath, bar soap)*Introduction*

Bath soap (bar soap) usually has low toxic potential and causes mild gastroenteritis with vomiting if ingested.

Treatment

To treat ingestion, include dilution with water, administration of intravenous fluids to maintain hydration, and administration of antiemetic and gastroprotectant drugs to treat gastroenteritis.

Sodium fluoroacetate (1080, 1081)*Introduction*

Sodium fluoroacetate is a colorless, odorless, tasteless compound that causes uncoupling of oxidative phosphorylation. The toxic dose in dogs and cats is 0.05-1.0 mg/kg. Clinical signs of toxicity include CNS excitation, seizures, and coma secondary to cerebral edema. The prognosis is guarded.

Treatment

To treat toxicity, procure and maintain a patent airway, monitor and stabilize the cardiovascular status, and control hyperthermia. Perform orogastric lavage, followed by administration of activated charcoal. If clinical signs are not present at the time of presentation, induce emesis. Administer intravenous fluids and supplemental oxygen, as necessary.

Strattera (selective norepinephrine reuptake inhibitor)*Introduction*

Strattera (atomoxetine hydrochloride) is a selective norepinephrine reuptake inhibitor used in the treatment of attention deficit hyperactivity disorder (ADHD) in humans. Peak serum concentrations occur in dogs within 3 to 4 hours of ingestion, with a peak half-life at 4 to 5 hours following ingestion. Clinical signs of toxicity include cardiac tachyarrhythmias, hypertension, disorientation, agitation, trembling, tremors, and hyperthermia.

Treatment

Treatment of intoxication is largely symptomatic and supportive in nature. First, induce emesis if the patient is conscious and has an intact gag reflex. Orogastric lavage can also be performed. Administer one dose of activated charcoal to prevent further absorption of the compound from the gastrointestinal tract. Identify cardiac dysrhythmias and treat accordingly. Control hypertension with sodium nitroprusside or diltiazem as a constant rate infusion. Administer acepromazine or chlorpromazine to control agitation. DO NOT use diazepam, because it can potentially worsen clinical signs. Administer intravenous fluids to maintain hydration and promote diuresis.

Strychnine*Introduction*

Strychnine is the active ingredient in pesticides used to control rodents and other vermin. The toxic dose in dogs is 0.75 mg/kg, and in cats is 2 mg/kg. Strychnine antagonizes spinal inhibitory neurotransmitters and causes severe muscle tremors, muscle rigidity, and seizures. Clinical signs are stimulated or exacerbated by noise, touch, light, and sound. Mydriasis, hyperthermia, and respiratory paralysis can occur. If strychnine toxicity is suspected, gastric contents should be collected and saved for analysis.

Treatment

If the animal is asymptomatic at the time of presentation, induce emesis. If clinical signs are present, perform orogastric lavage. Both emesis and orogastric lavage should be followed by the administration of activated charcoal. Administer intravenous crystalloid fluids to support the cardiovascular system, aid in cooling measures, and improve renal diuresis. Treat CNS stimulation with methocarbamol, diazepam, or phenobarbital. The animal should have cotton packed in its ears to prevent noise stimulation, and should be placed in a quiet, dark room.

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Styptic pencil*Introduction*

Styptic pencils contain potassium alum sulfate, a compound with a low toxic potential. Ingestion of styptic pencils is corrosive due to the release of sulfuric acid during hydrolysis of the salt.

Treatment

Treatment of ingestion includes dilution with Milk of Magnesia or water, administration of antiemetic and gastroprotectant drugs, and administration of intravenous crystalloid fluids to maintain hydration. Do NOT induce emesis, because of the risk of causing further esophageal irritation.

Sunscreen: see zinc and zinc oxide

Suntan lotion: see shampoos, zinc-based, and alcohols

Tar: see fuels

Tea tree oil (melaleuca oil)*Introduction*

Tea tree (melaleuca) oil is an herbal-origin flea-control product. The toxic principles in tea tree oil are monoterpenes, which produce clinical signs of neuromuscular weakness, and ataxia.

Treatment

Treatment of tea tree oil toxicity includes administration of cathartics and activated charcoal to prevent further absorption. Carefully bathe the animal to prevent further dermal exposure.

Tetanus*Introduction*

Tetanus spores from *Clostridium tetani* organisms are ubiquitous in the soil and feces, particularly in barnyards. Cases have been reported in dogs after tooth eruption and after abdominal surgeries performed with cold sterilization packs. Anaerobic wound infections can contain tetanus spores. The neurotoxin from *C. tetani* inhibits spinal inhibitory neurons, causing motor neuron excitation. Extensor muscle rigidity (“sawhorse stance”), erect ears, and risus sardonicus (a sardonic grin) are characteristic features of tetanus.

Treatment:

Administer tetanus antitoxin if toxin has not already been bound in the CNS. To eliminate the source of the toxin (e.g., abscess), open and debride all wounds. Intravenous administration of ampicillin or penicillin G is the treatment of choice for tetanus. Supportive care in the form of skeletal muscle relaxants, intravenous fluids and parenteral nutrition, and nursing care to prevent decubitus ulcer formation is required. In extreme cases, mechanical ventilation may be necessary.

Toilet bowl cleaners: see acids/corrosives

Triazenes*Introduction*

Triazene compounds include atrazine, prometon, and monuron (Telvar). The toxic mechanism of triazene compounds is unknown. Clinical signs of toxicity include salivation, ataxia, hyporeflexia, contact dermatitis, hepatorenal damage, muscle spasms, respiratory difficulty, and death.

Treatment

Treatment of triazene exposure includes cardiovascular and renal support in the form of intravenous crystalloid fluids, inotropic drugs, and antiarrhythmic agents, as necessary. If the exposure is recent, induce emesis. Perform orogastric lavage in animals that cannot

protect the airway. Emesis and orogastric lavage should be followed by the administration of activated charcoal and a cathartic. Carefully bathe the patient to prevent further dermal absorption.

Tricyclic antidepressants

Introduction

A variety of tricyclic antidepressants are available for use in both humans and animals, including amitriptyline, amoxapine, desipramine, doxepine, fluoxetine (Prozac), fluvoxamine (Luvox), imipramine, nortriptyline, paroxetine (Paxil), protriptyline, sertraline (Zoloft), and trimipramine. Selective serotonin reuptake inhibitors (SSRIs) are rapidly absorbed from the digestive tract, with peak serum concentrations occurring 2 to 8 hours after ingestion. The elimination half-life for each drug differs in dogs, but typically last 16 to 24 hours. SSRIs inhibit the reuptake of serotonin, causing serotonin to accumulate in the brain. This can cause “serotonin syndrome,” characterized by trembling, seizures, hyperthermia, ptialism or hypersalivation, cramping or abdominal pain, vomiting, and diarrhea. Other clinical signs of SSRI intoxication include depression, tremors, bradycardia, tachyarrhythmias, and anorexia. Any animal that has ingested an SSRI should be promptly treated and carefully observed for at least 72 hours for side effects.

Treatment

The treatment of suspected SSRI intoxication involves gastric decontamination if the patient is not depressed and has an intact gag reflex. Perform orogastric lavage and administer activated charcoal to prevent further toxin absorption and hasten elimination from the gastrointestinal tract. Treat other clinical signs symptomatically. Administer intravenous diazepam to control seizures. Treat tachyarrhythmias according to type. Administer methocarbamol to control muscle tremors. Cyproheptadine (1 mg/kg), a serotonin antagonist, can be dissolved in water and administered per rectum.

Turpentine: See Fuels

Vitamin K antagonist rodenticides

Introduction

Vitamin K antagonist rodenticides, which are commonly found in pelleted or block form, inhibit the activation of the vitamin K–dependent coagulation factors II, VII, IX, and X. Clinical signs of hemorrhage occur within 2 to 7 days of exposure. Hemorrhage can occur anywhere in the body, and can be manifested as petechiation of the skin or mucous membranes, hemorrhagic sclera, epistaxis, pulmonary parenchymal or pleural hemorrhage, gastrointestinal hemorrhage, pericardial hemorrhage, hematuria, retroperitoneal hemorrhage, hemarthrosis, and central nervous system hemorrhage. Clinical signs include respiratory distress, cough, bleeding from the gums or into the eyes, ataxia, paresis, paralysis, seizures, hematuria, joint swelling, lameness, lethargy, weakness, inappetence, and collapse.

Diagnosis is made based on clinical signs and a prolonged activated clotting time, or prothrombin time. The PIVKA (proteins induced by vitamin K antagonism) test may be helpful but usually cannot be performed in-house. Slight thrombocytopenia may be present secondary to hemorrhage; however, blood levels usually do not reach the critical level of $<50,000$ platelets/ μL to cause clinical signs of hemorrhage. In some cases, severe stress-induced hyperglycemia and glucosuria may be present but resolves within 24 hours.

Treatment

If the rodenticide was ingested within the last 2 hours, induce emesis. Alternatively, orogastric lavage can be performed in an uncooperative patient. Both emesis and orogastric lavage should be followed by administration of activated charcoal. The stomach contents can be submitted for analysis. Following successful treatment, administer oral vitamin K for 30 days after the exposure; or a check prothrombin time 2 days after gastric decontamination. If the prothrombin time is prolonged, administer fresh frozen plasma and Vitamin K.

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If the prothrombin time is normal, gastric decontamination was successful, and no further treatment is necessary.

If an animal presents with clinical signs of intoxication, administer activated clotting factors in the form of fresh frozen plasma (20 mL/kg), and vitamin K₁ (5 mg/kg SQ in multiple sites with a 24-gauge needle). Packed RBCs or fresh whole blood may be required if the patient is also anemic. Supportive care in the form of supplemental oxygen may be necessary in cases of pulmonary or pleural hemorrhage. Following initial therapy and discharge, the patient should receive vitamin K₁ (2.5 mg/kg PO q8-2h for 30 days), and prothrombin time should be checked 2 days after the last vitamin K capsule is administered. In some cases, depending on the type of anticoagulant ingested, an additional 2 weeks of vitamin K1 therapy may be required.

Window cleaner: see ethylene glycol

Xylitol

Introduction

Xylitol is a sugar alcohol that, when ingested by humans, does not cause a significant increase in blood glucose, and therefore does not stimulate insulin release from the human pancreas. In dogs, however, xylitol causes a massive rapid and dose-dependent release of insulin from pancreatic beta-cells. Following insulin release, clinically significant hypoglycemia can develop, followed by signs of vomiting, weakness, ataxia, mental depression, hypokalemia, hypoglycemic seizures, and coma. Clinical signs associated with xylitol ingestion can be seen within 30 minutes of ingestion and can last for more than 12 hours, even with aggressive treatment.

Treatment

Known xylitol ingestion should be treated as for other toxin ingestion. If no neurologic abnormalities exist at the time the patient is seen, induce emesis, followed by administration of activated charcoal. It remains unknown at this time whether activated charcoal actually delays or prevents the absorption of xylitol from the canine gastrointestinal tract. If clinical signs have already developed, perform orogastric lavage and gastric decontamination. Blood glucose concentrations should be analyzed and maintained with supplemental dextrose as a constant rate infusion (2.5%-5%) until normoglycemia can be maintained with multiple frequent small meals. Hypokalemia may develop because it is driven intracellularly by the actions of insulin. Treat hypokalemia with supplemental potassium chloride by infusion, not to exceed 0.5 mEq/kg/hour.

Zephiran: see detergents, cationic

Zinc and zinc oxide

Introduction

Pennies minted in the U.S. after 1982 contain large amounts of zinc rather than copper. Other sources of zinc include zinc oxide ointment and hardware such as that found in metal bird cages. Zinc toxicity causes intravascular hemolysis, anemia, gastroenteritis, and renal failure.

Treatment

If zinc toxicity is suspected, take an abdominal radiograph to document the presence of the metal in the stomach or intestines. (If zinc-containing ointment was ingested, this will not be visible on radiographs.) Induce emesis or perform orogastric lavage, depending on the size of the object ingested. Often, small objects such as pennies can be retrieved using endoscopy or surgical gastrotomy/enterotomy. Always take an additional radiograph after the removal procedure to ensure that all objects have been successfully removed. Administer intravenous fluids to maintain renal perfusion and promote fluid diuresis. Administer gastroprotectant and antiemetic drugs. Chelation therapy with succimer, calcium EDTA, dimercaprol, or penicillamine may be necessary. Do NOT administer

calcium EDTA if the patient is dehydrated, because renal failure can result. Severe anemia should be treated with packed RBCs or hemoglobin-based oxygen carriers.

Additional Reading

- Cope RB: Four new small animal toxicoses. *Aust Vet Pract* 34(3):121-123, 2004.
- Donaldson CW: Paintball toxicosis in dogs. *Vet Med* 98(12):995-998, 2003.
- Dunayer ER: Hypoglycemia following canine ingestion of xylitol-containing gum. *Vet Hum Toxicol* 46(2):87-88, 2004.
- Gfeller RW, Messonnier SP: *Handbook of small animal toxicology and poisonings*, ed 2, St. Louis, 2004, Mosby.
- Hansen SR: Macadamia nut toxicosis in dogs. *Vet Med* 97(4):274-276, 2002.
- Hopper K, Aldrich J, Haskins S: The recognition and treatment of the intermediate syndrome of organophosphate poisoning in a dog. *J Vet Emerg Crit Care* 12(2):99-103, 2002.
- Mazzaferro EM, Eubig PA, Hackett TB et al: Acute renal failure in four dogs after raisin or grape ingestion (1999-2002). *J Vet Emerg Crit Care* 14(3):203-212, 2004.
- Plum Lee KH: *Clinical veterinary toxicology*. Mosby, St. Louis, 2004.
- Roder JD: *Veterinary toxicology*. Butterworth-Heinemann, Woburn, Mass, 2001.

RESPIRATORY EMERGENCIES

Respiratory emergencies consist of any problem that impairs delivery of oxygen to the level of the alveoli or diffusion of oxygen across the alveolar capillary membrane into the pulmonary capillary network. Decreased respiratory rate or tidal volume can result in hypoxia and buildup of carbon dioxide, or hypercarbia, leading to respiratory acidosis. Conditions most frequently encountered result in airflow obstruction, prevention of normal lung expansion, interference with pulmonary gas exchange (ventilation-perfusion mismatch), and alterations of pulmonary circulation. Evaluation of the patient with respiratory distress is often challenging, because the most minimal stress can cause rapid deterioration, or even death in critical cases. Careful observation of the patient from a distance often allows the clinician to determine the severity of respiratory distress and localize the lesion based on the patient's respiratory pattern and effort.

Animals in respiratory distress often have a rapid respiratory rate (>30 breaths per minute). As respiratory distress progresses, the patient may appear anxious and start open-mouth breathing. The animal often develops an orthopneic posture, characterized by neck extension, open-mouthed breathing, and elbows abducted or pulled away from the body. Cyanosis of the mucous membranes often indicates extreme decompensation. Clinical signs of respiratory distress can develop acutely, or from decompensation of a more chronic problem that was preceded by a cough, noisy respirations, or exercise intolerance.

Localization of the cause of respiratory distress is essential to successful case management. In any patient with clinical signs of respiratory distress, the differential diagnosis should include primary pulmonary parenchymal disease, airway disease, thoracic cage disorders, congestive heart failure, dyshemoglobinemias (carbon monoxide, methemoglobin), and anemia. Careful observation of the patient's respiratory pattern can aid in making a diagnosis of upper airway disease/obstruction, primary pulmonary parenchymal disease, pleural space disease, and abnormalities of the thoracic cage. It is often helpful to rest a hand on the patient and breathe along with the patient's effort, to confirm the periods of inhalation and exhalation.

The pharynx, larynx, and extrathoracic trachea comprise the upper airway. Obstructive lesions are associated with a marked inspiratory wheeze or stridor and slow deep inspiratory effort. Auscultation of the larynx and trachea may reveal more subtle obstructions of normal air flow. Stridor can usually be auscultated without the use of a stethoscope. Lung sounds are usually normal. The neck should be carefully palpated for a mass lesion, tracheal collapse, and subcutaneous emphysema. Subcutaneous emphysema suggests tracheal damage or collapse secondary to severe trauma. In some cases, there is a history of voice, or bark, change secondary to laryngeal dysfunction. Differential diagnosis is usually based on the patient's signalment, history, and index of suspicion of a particular disease process. Differential diagnoses of upper airway obstruction are listed in Box 1-61.

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BOX 1-61 DIFFERENTIAL DIAGNOSES OF UPPER AIRWAY OBSTRUCTION

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| <ul style="list-style-type: none"> • Abscess • Brachycephalic airway syndrome • Granuloma • Laryngeal collapse • Laryngeal paralysis • Nasopharyngeal polyp | <ul style="list-style-type: none"> • Neoplasia • Obstructive laryngitis • Pharyngeal foreign body • Tracheal collapse • Tracheal foreign body • Traumatic fracture of larynx or tracheal cartilage |
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Diseases of the pleural space often are associated with a restrictive respiratory pattern. Inspiratory efforts are short, rapid, and shallow, and there is often a marked abdominal push. The pattern has been referred to as a choppy “dysynchronous” respiratory pattern. Depending on the disease present, lung sounds may be muffled ventrally and enhanced dorsally. Percussion of the thorax reveals decreased resonance if fluid is present. Increased resonance is present with pneumothorax. Decreased compressibility of the anterior thorax may be present with an anterior mediastinal mass lesion, particularly in cats and ferrets. A pneumothorax or diaphragmatic hernia is commonly associated with evidence of trauma, with or without rib fractures. Respiratory distress due to hemothorax may be exacerbated by anemia. Differential diagnoses for patients with evidence of pleural cavity disease include pneumothorax, diaphragmatic hernia, neoplasia, and various types of pleural effusion.

Primary pulmonary parenchymal disease can involve the intrathoracic airways, alveoli, interstitial space, and pulmonary vasculature. A rapid, shallow, restrictive respiratory pattern may be observed with a marked push on exhalation, particularly with obstructive airway disease such as chronic bronchitis (asthma) in cats. Crackles or wheezes are heard on thoracic auscultation. Differential diagnoses for pulmonary parenchymal disease include cardiogenic and noncardiogenic pulmonary edema, pneumonia, feline bronchitis (asthma), pulmonary contusion, aspiration pneumonitis, pulmonary thromboembolism, neoplasia, infection (bacterial, fungal, protozoal, viral), and/or chronic bronchitis.

Other abnormal respiratory patterns may be evident, and warrant further consideration. Tachypnea present in the absence of other signs of respiratory distress can be a normal response to nonrespiratory problems, including pain, hyperthermia, and stress. A restrictive respiratory pattern with minimal thoracic excursions can be associated with diseases of neuromuscular function, including ascending polyradiculoneuritis, botulism, and tick paralysis. If adequate ventilation cannot be maintained by the patient, mechanical ventilation may be indicated. Kussmaul respiration manifests as very slow, very deep respirations when a metabolic acidosis is present. This type of respiratory pattern typically is observed in patients with severe diabetic ketoacidosis and renal failure in a compensatory attempt to blow off carbon dioxide. Cheyne-Stokes respiration is usually observed with a defect in the central respiratory control center. The classic pattern of Cheyne-Stokes respiration is normal or hyperventilation followed by a period of apnea or hypoventilation. In cases of lower cervical cord damage or damage to the central respiratory control center in the CNS, the diaphragm alone may assume most of the ventilatory movement. With diaphragmatic fatigue, severe hypoventilation and resultant hypoxemia may require mechanical ventilation.

IMMEDIATE MANAGEMENT

Immediate management of any patient in respiratory distress is to minimize stress at all costs. Relatively benign procedures such as radiography or intravenous catheter placement can be fatal in patients with severe respiratory compromise. Stabilization should always precede further diagnostic evaluation. In some cases, sedation may be required before performing any diagnostics, to prevent further stress. All patients should receive some form of supplemental oxygen, either by mask, cage, or flow-by techniques. In cases in which a severe pneumothorax or pleural effusion is suspected, perform therapeutic and diagnostic thoracocentesis bilaterally to allow lung re-expansion and alleviate respiratory distress, whenever possible. If thoracocentesis alone is not effective at maintaining lung re-expansion,

place a thoracostomy tube (particularly in cases of tension pneumothorax). If hypovolemic/hemorrhagic shock is present, initiate treatment while stabilizing the respiratory system (see section on Shock).

If an animal is suspected of having an upper airway obstruction, reestablish airflow. In cases of laryngeal paralysis, tracheal collapse, and brachycephalic airway syndrome, sedation is often very useful in alleviating the distress of airway obstruction. In cases of laryngeal collapse, however, sedation may make the condition worse. If laryngeal edema is severe, administer a dose of short-acting glucocorticosteroids (dexamethasone sodium phosphate) to decrease laryngeal inflammation and edema. If a foreign body is lodged in the pharynx, perform the Heimlich maneuver by thrusting bluntly several times on the patient's sternum. Objects such as balls or bones may be small enough to enter the larynx but too large to be expelled, and will require rapid-acting general anesthesia to facilitate dislodgement and removal. If the obstruction cannot be removed, bypassing the obstruction with an endotracheal tube or temporary tracheostomy should be considered.

In an emergency, a temporary transtracheal oxygen catheter can quickly be placed in the following manner. Connect a 20- or 22-gauge needle to a length of intravenous extension tubing and a 3-mL syringe. Place the male connector of the syringe into the female portion of the extension tubing. Cut off the syringe plunger and connect the resulting blunt end to a length of flexible tubing attached to a humidified oxygen source. Run the oxygen at 10 L/minute to provide adequate oxygenation until a tracheostomy can be performed. (See sections on Oxygen Supplementation and Tracheostomy).

Once the animal's condition has been stabilized, specific diagnostic tests, including arterial blood gas analyses, thoracic radiographs, and/or transtracheal wash, can be performed, depending on the patient's condition and needs. Specific therapies for management of upper airway obstruction, pleural space disease, and pulmonary disease are discussed next.

Management of upper airway obstruction

Upper airway obstruction can occur as a result of intraluminal or extraluminal mass lesions or foreign bodies in the oropharynx (abscess, neoplasia), laryngeal paralysis, trauma, and anatomic abnormalities. Clinical signs of an upper airway obstruction are associated with an animal's extreme efforts to inhale air past the obstruction. Marked negative pressure occurs in the extrathoracic airways and can cause worsening of clinical signs. Mucosal edema and inflammation further worsen the obstruction.

Therapy for upper airway obstruction is aimed at breaking the cycle of anxiety and respiratory distress. Administer the anxiolytic tranquilizer acepromazine (0.02-0.05 mg/kg IV, IM, SQ) to decrease patient anxiety. Many animals develop hyperthermia from increased respiratory effort and extreme anxiety. Implement cooling measures in the form of cool intravenous fluids and wet towels soaked in tepid water placed over the animal (see section on Hyperthermia). Administer supplemental oxygen in a manner that is least stressful for the animal. Short-acting glucocorticosteroids can also be administered (dexamethasone sodium phosphate, 0.25 mg/kg IV, SQ, IM) to decrease edema and inflammation.

If the airway obstruction is severe and there is no response to initial measures to alleviate anxiety and decrease inflammation, establish control of ventilation by placement of an endotracheal tube (see section on Endotracheal Intubation), tracheal oxygen catheter, or temporary tracheostomy. To obtain airway control, administer a rapid-acting anesthetic (propofol, 4-7 mg/kg IV to effect), and intubate with a temporary tracheostomy. An intra-tracheal oxygen catheter can be placed with sedation and/or a local anesthetic (see technique for transtracheal wash).

LARYNGEAL PARALYSIS

Laryngeal paralysis is a congenital or acquired condition that occurs primarily in large-breed dogs secondary to denervation of the arytenoid cartilages by the recurrent laryngeal nerve. Congenital laryngeal paralysis occurs in the Bouvier des Flandres, Siberian Husky, and Bull Terrier. Acquired laryngeal paralysis occurs in Labrador Retrievers, Saint Bernards,

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and Irish Setters. Acquired laryngeal paralysis can be idiopathic, acquired secondary to trauma to the recurrent laryngeal nerve, or can be a component of systemic neuromuscular disease. Although rare, this condition also occurs in cats.

With dysfunction of the recurrent laryngeal nerve, the intrinsic laryngeal muscles atrophy and degenerate. As a result, the vocal folds and arytenoid cartilage move in a paramedian position within the airway and fail to abduct during inhalation, causing airway obstruction. Laryngeal paralysis can be partial or complete, unilateral or bilateral. In many cases, a change in bark is noted prior to the development of clinical signs of respiratory distress or exercise intolerance. When a patient presents with severe inspiratory stridor (with or without hyperthermia) initiate stabilization with anxiolytic tranquilizers, supplemental oxygen, and cooling measures. Once the patient's condition has been stabilized, definitive measures to accurately document and assess the patient's airway should be considered. Place the patient under very heavy sedation with short-acting barbiturates or propofol (4–7 mg/kg IV) and observe the arytenoid cartilages closely in all phases of respiration. Administer just enough drug to allow careful examination without getting bitten. If the arytenoid cartilages do not abduct during inhalation, administer Dopram (doxapram hydrochloride, 1–5 mg/kg IV) to stimulate respiration.

Absent or paradoxical laryngeal motion (closed during inspiration and open during exhalation) is characteristic of laryngeal paralysis. Correction of the defect involves documentation and treatment of any underlying disorder and surgical repair of the area to open the airway. Partial laryngectomy, arytenoid lateralization (“tie-back” surgery), or removal of the vocal folds has been used with some success. Aspiration pneumonia is common following these procedures.

BRACHYCEPHALIC AIRWAY SYNDROME AND LARYNGEAL COLLAPSE

Brachycephalic airway syndrome is associated with a series of anatomic abnormalities that collectively increase resistance to airflow. Affected animals typically have stenotic nares, an elongated soft palate, and a hypoplastic trachea. Components of the syndrome can occur alone or in combination. In severe cases, laryngeal saccular edema and eversion, and eventual pharyngeal collapse, can occur secondary to the severe increase in intrathoracic airway pressure required to overcome the resistance of the upper airways. Specific airway anomalies can be identified with general anesthesia and laryngoscopy.

Severe respiratory distress should be treated as discussed previously. Treatment requires surgical correction of the anatomic abnormalities. In animals with laryngeal collapse, surgical correction may not be possible, and a permanent tracheostomy may be required. Because an elongated soft palate and stenotic nares can be identified before the onset of clinical signs, surgical correction to improve airflow when the animal is young may decrease the negative intra-thoracic pressure necessary to move air past these obstructions. The chronic consequences of everted laryngeal sacs and laryngeal collapse potentially can be prevented.

TRACHEAL COLLAPSE

Tracheal collapse is common in middle-aged and older toy and small-breed dogs. The owner typically reports a chronic cough that is readily induced by excitement or palpation of the trachea. The cough often sounds like a “goose honk.” Diagnostic confirmation is obtained by lateral radiography or fluoroscopy of the cervical and thoracic trachea during all phases of respiration. Acute decompensation is uncommon but does occur, particularly with excitement, exercise, and increased environmental temperatures or ambient humidity.

Therapy of the patient with acute respiratory distress secondary to tracheal collapse includes sedation, administration of supplemental oxygen, and provision of cooling measures to treat hyperthermia. Cough suppressants (hydrocodone bitartrate–homatropine methylbromide, 0.25 mg/kg PO q8–12h, or butorphanol, 0.5 mg/kg PO q6–12h) are useful. Tracheal collapse is a dynamic process that usually involves both the upper and lower airways. Because of this, bypassing the obstruction is often difficult. Tracheal stents have been

used with limited success in combination with treatment of chronic lower airway disease.

TRAUMA

Crush or bite injuries to the neck can result in fractures or avulsion of the laryngeal or tracheal cartilages. Bypassing the obstructed area may be necessary until the patient is stable and can undergo surgical correction of the injury. If there is avulsion of the cranial trachea, it may be difficult to intubate the patient. A long, rigid urinary catheter can be inserted past the area of avulsion into the distal segment, and an endotracheal tube passed over the rigid catheter, to establish a secure airway. Neck injury can also result in damage to the recurrent laryngeal nerve and laryngeal paralysis.

FOREIGN BODIES

Foreign bodies can lodge in the nasal cavity, pharynx, larynx, and distal trachea. Signs of foreign bodies in the nares include acute sneezing and pawing at or rubbing the muzzle on the ground. If the object is not removed, sneezing continues and a chronic nasal discharge develops. Respiratory distress is uncommon, but the foreign body is severely irritating. Pharyngeal and tracheal foreign bodies can cause severe obstruction to airflow and respiratory distress. Diagnosis of a foreign body is based on the patient history, physical examination findings, and thoracic or cervical radiographs. Smaller foreign bodies lodged in the distal airways may not be apparent radiographically but can cause pulmonary atelectasis.

Foreign bodies of the nose or pharynx can often be removed with an alligator forceps with the patient under anesthesia. If removal is not possible with a forceps, flushing the nasal cavity from cranial to caudal (pack the back of the mouth with gauze to prevent aspiration) can sometimes dislodge the foreign material into the gauze packing. Rhinoscopy may be necessary. If an endoscope is not available, an otoscope can be used.

Foreign objects lodged in the trachea can be small and function like a ball valve during inhalation and exhalation, causing episodic hypoxia and collapse. When attempting to remove these objects, suspend the patient with its head down. Remove the object with an alligator forceps, using a laryngoscope to aid in visualization. Foreign bodies lodged in the trachea or bronchi require removal with endoscopic assistance.

INTRALUMINAL MASSES

Nasopharyngeal polyps (in cats, tumors, obstructive laryngitis, granulomas, abscesses, and cysts) can cause upper airway obstruction. Clinical signs are usually gradual in onset. The lesions can be identified through careful laryngoscopic examination performed with the patient under general anesthesia. The nasopharynx above the soft palate should always be included in the examination. Pedunculated masses and cysts are excised at the time of evaluation. Biopsy of diffusely infiltrative masses is indicated for histologic examination and prognosis. It is impossible to distinguish obstructive laryngitis from neoplasia based on gross appearance alone. Whenever possible, material should be collected from abscesses and granulomas for cytologic evaluation and bacterial culture.

EXTRALUMINAL MASSES

Extraluminal masses impinge on and slowly compress the upper airways, resulting in slow progression of clinical signs. Masses are usually identified by palpation of the neck. Enlarged mandibular lymph nodes, thyroid tumors, and other neoplasms may be present. Diagnosis is usually based on a combination of radiography and ultrasonography. CT and/or MRI are helpful in identifying the full extent and invasiveness of the lesion. Definitive diagnosis is made with a fine-needle aspirate or biopsy. Many thyroid tumors bleed excessively.

PLEURAL CAVITY DISEASE

The inside of each side of the hemithorax is covered in parietal pleura. The lung lobes are covered in visceral pleura. The two surfaces are in close contact with each other, and are

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contiguous at the hilum under normal circumstances. Pneumothorax refers to free air within the pleural space, accumulating in between the parietal and visceral pleura. The term *pleural effusion* refers to fluid accumulation in that area but does not reflect the amount or type of fluid present. The mediastinal reflections of the pleura typically are thin in dogs and cats, and usually, but not always, connect. Bilateral involvement of pneumothorax or pleural effusion is common. Both pneumothorax and pleural effusion compromise the lungs' ability to expand and result in hypoxia and respiratory distress.

Pneumothorax

Pneumothorax can be classified as open versus closed, simple versus complicated, and tension. An open pneumothorax communicates with the external environment through a rent in the thoracic wall. A closed pneumothorax results from tears in the visceral pleura but does not communicate with the outside. A tension pneumothorax occurs as a result of a tear in the lung or chest wall that creates a flap valve, such that air is allowed to leave the lung and accumulate in the pleural space during inhalation, and closes to seal off exit of air from the pleural space during exhalation. Tension pneumothorax can cause rapid decline in cardiopulmonary status and death if not recognized and treated immediately. A simple pneumothorax is one that can be controlled with a simple thoracocentesis. Complicated pneumothorax involves repeated accumulation of air, requiring placement of a thoracic drainage catheter.

In many cases, pneumothorax develops as a result of trauma. Spontaneous pneumothorax occurs with rupture of cavitory lesions of the lung that may be congenital or acquired as a result of prior trauma, heartworm disease, airway disease (emphysema), paragonimiasis, neoplasia, or lung abscess. Pneumothorax also rarely occurs as a result of esophageal tears or esophageal foreign bodies.

Rapid circulatory and respiratory compromise following traumatic pneumothorax can develop as a result of open or tension pneumothorax, rib fractures, airway obstruction, pulmonary contusions, hemothorax, cardiac dysrhythmias, cardiac tamponade, and hypovolemic shock. Any patient that is rapidly decompensating after a traumatic episode must be quickly assessed, and emergency therapy initiated (see section on Immediate Management of Trauma, a CRASH plan).

Diagnosis of pneumothorax is usually made based on a history of trauma, a rapid, shallow, restrictive respiratory pattern, and muffled heart and lung sounds on thoracic auscultation. The clinical signs and history alone should prompt the clinician to perform a bilateral diagnostic and therapeutic thoracocentesis before taking thoracic radiographs (see section on Thoracocentesis). The stress of handling the patient for radiography can be deadly in severe cases of pneumothorax. Although the mediastinum on both sides of the thorax connects, it is necessary to perform thoracocentesis on both sides to ensure maximal removal of free air in the pleural space and allow maximal lung expansion. If negative pressure cannot be obtained, or if the patient rapidly reaccumulates air, place a thoracostomy tube connected to continuous suction. (See section on Thoracostomy Tube Placement).

Management of open sucking chest wounds in pneumothorax

Treat all penetrating wounds to the thorax as open sucking chest wounds unless proved otherwise. To "close" an open sucking chest wound, clip the fur around the wound as quickly as possible, and place sterile lubricant jelly or antimicrobial ointment circumferentially around the wound. Cut a sterile glove to provide a covering. Place the covering over the wound, making sure to cover all of the sterile lubricant, thus creating a seal to close the wound temporarily from the external environment. Evaluate the patient's thorax via thoracocentesis while placing a thoracostomy tube. Once the patient is stable, the open chest wound can be surgically explored, lavaged, and definitively corrected. All animals with open chest wounds should receive antibiotics (first-generation cephalosporin) to prevent infection. Following stabilization, radiographs can be taken and evaluated. Pneumothorax is confirmed by evidence of elevation of the cardiac silhouette above the sternum, increased density of the pulmonary parenchymal tissue, free air in between the parietal and visceral

pleura (making the outline of the lungs visible), and absence of pulmonary vascular structures in the periphery. Parenchymal lesions within the lungs are best identified after as much air as possible has been removed from the thorax. Obtain left and right lateral and ventrodorsal or dorsoventral views. A standing lateral view may reveal air- or fluid-filled cavitory masses. If underlying pulmonary disease is suspected as a cause of spontaneous pneumothorax, a transtracheal wash, fecal flotation, and heartworm test may be indicated.

Treatment of pneumothorax

Treatment of pneumothorax includes immediate bilateral thoracocentesis, covering of any open chest wounds, administration of supplemental oxygen, and placement of a thoracostomy tube if negative pressure cannot be obtained or if air rapidly reaccumulates. Serial radiography, CT, or MRI should be performed in dogs with spontaneous pneumothorax, because the condition can be associated with generalized pulmonary parenchymal disease. Strict cage rest is required until air stops accumulating and the thoracostomy tube can be removed. The patient's chest tube should be aspirated every 4 hours after discontinuing continuous suction. If no air reaccumulates after 24 hours, the chest tube can be removed. Exercise restriction is indicated for a minimum of 1 week. If bullae or mass lesions are present, exploratory thoracotomy should be considered as a diagnostic and potentially therapeutic option for long-term management in prevention of recurrence.

Pleural effusion

Pleural fluid cytologic analysis is indicated for all patients with pleural effusion before administration of antibiotics. The general term *pleural effusion* means a collection of fluid in the space between the parietal and visceral pleura but does not indicate what kind or how much fluid is present. Clinical signs associated with pleural effusion depend on how much fluid is present, and how rapidly the fluid has accumulated. Clinical signs associated with pleural effusion include respiratory distress, reluctance to lie down, labored breathing with an abdominal component on exhalation, cough, and lethargy. Auscultation of the thorax may reveal muffled heart and lung sounds ventrally and increased lung sounds dorsally, although pockets of fluid may be present, depending on the chronicity of the effusion. Percussion of the thorax may reveal decreased resonance.

In stable patients, the presence of pleural effusion can be confirmed radiographically. Radiographic confirmation of the pleural effusion should include right and left lateral and dorsoventral or ventrodorsal views. A handling or standing lateral view should be obtained if an anterior mediastinal mass is suspected. The standing lateral view will allow the fluid to collect in the costophrenic recess.

In patients with respiratory distress, muffled heart and lung sounds, and suspicion of pleural effusion, thoracocentesis should be performed immediately. Thoracocentesis can be both therapeutic and diagnostic. Radiography is contraindicated because the procedure can cause undue stress and exacerbation of clinical signs in an unstable patient. Pleural effusion can cause severe respiratory distress, and can be the result of a number of factors that must be considered when implementing an appropriate treatment plan. Pathology of the pleura is almost always a secondary process except for primary bacterial pleuritis and pleural mesotheliomas. Causes of pleural effusion in the cat and dog include pyothorax, feline infectious peritonitis, congestive heart failure, chylothorax, heartworm disease, hemothorax, hypoalbuminemia, lung lobe torsions, neoplasia, diaphragmatic hernia, and pancreatitis (Box 1-62). In stable animals, diagnosis of pleural effusion can be made based

BOX 1-62 PHYSIOLOGIC PROCESSES ASSOCIATED WITH PLEURAL EFFUSION

- Imbalance of transpleural or hydrostatic or protein osmotic forces
- Change in membrane permeability
- Decrease in rate of fluid reabsorption
- Combination of foregoing mechanisms

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on thoracic radiography or ultrasound. Thoracic radiographs can show whether the pleural effusion is unilateral or bilateral. Effusions in dogs and cats are usually bilateral. The lung parenchyma and the cardiac silhouette cannot be fully evaluated until most of the fluid has been evacuated from the pleural cavity. Following thoracocentesis, radiography should be performed with left and right lateral and ventrodorsal or dorsoventral views. In cases of suspected heart failure, echocardiography also is necessary.

Pleural fluid cytologic analysis is indicated for all patients with pleural effusion. Collect specimens before administering antibiotics, whenever possible, because treatment with antibiotics can make a septic condition (pyothorax) appear nonseptic. The remainder of the diagnostic workup and treatment is based on the type of fluid present (Table 1-52). The fluid may be a transudate, nonseptic exudate, septic exudate, chylous, hemorrhagic, or neoplastic. Ultrasonographic evaluation of the thorax can be helpful in identifying intrathoracic masses, diaphragmatic hernias, lung lobe torsions, and cardiac abnormalities. Unlike radiography, ultrasonography is facilitated by the presence of fluid in the pleural space.

Pyothorax

Pyothorax refers to a septic effusion of the pleural cavity. The infection is generally the result of a combination of aerobic and anaerobic bacteria. Rarely, fungal organisms are present. The source of the underlying organisms is rarely identified, particularly in cats, but can be caused by penetrating wounds through the chest wall, esophagus, migrating foreign bodies (especially grass awns), or primary lung infections. The most common organisms associated with pyothorax in the cat are *Pasteurella*, *Bacteroides*, and *Fusobacterium*. Fever is often present in addition to clinical signs of pleural effusion. Septic shock is uncommon.

Diagnosis of pyothorax is made based on cytologic analysis and the demonstration of intracellular and extracellular bacteria, toxin neutrophils and macrophages, and sometimes the presence of sulfur granules. Gram stains of the fluid can assist in the initial identification of some organisms. Bacterial cultures are indicated for bacteria identification and antibiotic susceptibility testing. Administration of antibiotics before cytologic evaluation can cause a septic effusion to appear nonseptic.

Emergency treatment for pyothorax involves placement of an intravenous catheter, intravenous fluids to treat hypovolemic shock, and broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6h, and enrofloxacin, 10 mg/kg IV q24h). Chloramphenicol also is an appropriate antibiotic to use for penetration into pockets of fluid. Administration of a beta-lactam antibiotic (ampicillin or amoxicillin) with a beta-lactamase inhibitor (amoxicillin clavulanate or ampicillin sulbactam) is helpful in achieving better coverage of *Bacteroides* spp.

Treatment of pyothorax differs in the cat and dog. In the cat, placement of one or two thoracic drainage catheters is recommended to allow continuous drainage of the intrathoracic abscess. Inadequate drainage can result in treatment failure. Fluid should be evaluated and the pleural cavity lavaged with 10 mL/kg of warmed 0.9% saline or lactated Ringer's solution every 8 hours. Approximately 75% of the infused volume should be recovered after each lavage.

In dogs, or in cats with refractory pyothorax, perform an exploratory thoracotomy to remove any nidus of infection. Rarely a foreign body is visible that can be removed at the time of surgery, but this finding is rare. Antibiotics are indicated for a minimum of 6 to 8 weeks after removal of the thoracostomy tube. Early diagnosis and aggressive treatment result in a good prognosis in the majority of patients with pyothorax. In cats, clinical signs of ptialism and hypothermia at the time of presentation worsen the prognosis.

Chylothorax

Chylothorax refers to the abnormal accumulation of chyle (lymphatic fluid) in the pleural cavity. The cisterna chili is the dilated collection pool of lymphatic ducts in the abdomen that accumulate chyle prior to entry into the thoracic duct located within the thoracic cavity.

TABLE 1 - 5.2 Analysis of Pleural Effusions

	Exudates				Hemorrhagic effusions
	Transudates	Modified transudates	Nonseptic exudates	Septic exudates	Chylous effusions
Color	Pale yellow	Yellow-pink	Yellow-pink	Yellow	White-pink
Transparency	Clear	Clear to cloudy	Cloudy	Cloudy to flocculent	Opaque
Protein (g/dL)	<2.5	<3.5	>3.0	>3.0	>2.5
RBCs	Absent to rare	Variable	Variable	Variable	Variable
Nucleated cells/mL	<500	<5000	>5000	>5000	400-10,000
Neutrophils	Rare	Variable number	Moderate	Moderate to high number	Acute: low number
Lymphocytes	Rare	Nondegenerative	Nondegenerative	Nondegenerative to degenerative	Chronic: moderate number
Macrophages	Occasional	Variable	Variable	Variable	Nondegenerative
Mesothelial cells	Occasional	Variable	Increased number	Increased number	Acute: high number
Fibrin	Absent	Occasional	Contain ingested debris	Rare	Chronic: low number
		Absent	Present	Present	Present
					Chronic: present
					Variable

Continued

TABLE 1 - 5.2 Analysis of Pleural Effusions—cont'd

	Exudates				Hemorrhagic effusions
	Transudates	Modified transudates	Nonseptic exudates	Septic exudates	
Bacteria	Absent	Absent	Absent	Present intra-and extracellularly	Absent
Lipid	Absent	Absent	Absent	Absent	Absent
Etiology	Right heart failure Hypoproteinemia	Chronic transudates Diaphragmatic hernia Neoplasia Right heart failure Pericardial disease	Neoplasia Feline infectious peritonitis Chronic diaphragmatic hernia Lung torsions Pyothorax	Foreign body Penetrating wound Idiopathic pyothorax	Trauma Neoplasia Bleeding disorders Lung torsions
				High triglycerides relative to low serum cholesterol; positive to lipotrophic stains	
				Idiopathic Congenital Lymphangiectasia Trauma Neoplasia Cardiac disease Pericardial disease Dirofilariasis	

The thoracic duct enters the thorax at the aortic hiatus. Numerous tributaries or collateral ducts exist. The functions of the lymphatic vessels collectively serve to deliver triglycerides and fat-soluble vitamins into the peripheral vascular circulation. Damage of the thoracic duct or lymphatic system or obstruction to lymphatic flow can result in the development of chylous effusion in the pleural or peritoneal space.

It is difficult to identify chylous effusions based on their milky appearance alone. To identify a chylous effusion versus a pseudo-chylous effusion, the triglyceride and cholesterol levels of the fluid must be compared with those of peripheral blood. Chylous effusions have a higher triglyceride and lower cholesterol levels than peripheral blood. Pseudo-chylous effusions have a higher cholesterol and lower triglyceride levels than peripheral blood.

Disease processes that can result in chylous effusions are listed in the Box 1-63. Clinical signs associated with chylous effusion are typical of any pleural effusion and of the disease process that caused the effusion. Weight loss may be evident, depending on the chronicity of the process.

The diagnosis is made based on thoracocentesis, cytology, and biochemical evaluation of the fluid (i.e., triglyceride and cholesterol levels). The fluid often appears milky or blood-tinged but can be clear if the patient has significant anorexia. Typical cytologic characteristics are listed in Table 1-52. Lymphangiography can be used to confirm trauma to the thoracic duct, but this is usually not necessary unless surgical ligation is going to be attempted. The diagnostic evaluation must also attempt to identify an underlying cause.

Therapy for chylothorax is difficult and primarily involves documentation and treatment of the underlying cause. If an underlying cause is not found, treatment is largely supportive and consists of intermittent thoracocentesis to drain the fluid as it accumulates and causes respiratory dysfunction, nutritional support, and maintenance of fluid balance. A variety of surgical techniques, including ligation of the thoracic duct, pleural-peritoneal shunts, and pleurodesis, have been attempted but have had limited success. Most recently, the combination of thoracic duct ligation with subtotal pericardectomy has been shown to improve surgical success rates in the treatment of chylothorax. Rutin, a bioflavonoid, has been used with limited success in the treatment of idiopathic chylothorax in cats. Prognosis in many cases of chylothorax is guarded.

Hemothorax

Extensive hemorrhage into the pleural cavity can cause fulminant respiratory distress due to sudden hypovolemia and anemia and interference with lung expansion. Hemothorax typically is associated with trauma, systemic coagulopathy, lung lobe torsions, and erosive lesions within the thorax (usually neoplasia). Diagnosis of hemothorax involves obtaining a fluid sample via thoracocentesis. Hemorrhagic effusion must be differentiated from systemic blood inadvertently collected during the thoracocentesis procedure. Unless the hemorrhage is peracute, fluid in cases of hemothorax is rapidly defibrinated and will not clot, has a packed cell volume less than that of venous blood, contains RBCs and macrophages. Hemorrhagic effusions also usually contain a disproportionately higher number of white blood cells compared with peripheral blood.

Hemothorax commonly is the sole clinical sign observed in animals with vitamin K antagonist rodenticide intoxication and systemic coagulopathy. Whenever an animal presents

BOX 1-63 CAUSES OF CHYLOUS EFFUSION

- | | |
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| <ul style="list-style-type: none"> • Cardiac disease • Diaphragmatic hernia • Heartworm disease • Idiopathic • Immune-mediated lymphadenitis • Lung lobe torsion | <ul style="list-style-type: none"> • Pericardial disease • Thoracic duct rupture • Thoracic lymphangiectasia • Thoracic neoplasia • Trauma • Venous thrombi |
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with signs of a hemorrhagic pleural effusion, perform coagulation testing immediately to determine whether a coagulopathy exists. The prothrombin time test is fast and can be performed as a cage-side test (see section on Coagulopathy).

Therapy for hemorrhagic pleural effusions should address the blood and fluid loss. Administer intravenous crystalloid fluids and RBC products (see section on Transfusion Therapy). When necessary, administer coagulation factors in the form of fresh whole blood or fresh frozen plasma, along with Vitamin K₁ (5 mg/kg SQ in multiple sites with a 25-gauge needle). If severe respiratory distress is present, evacuate the blood within the pleural space via thoracocentesis until clinical signs of respiratory distress resolve. Fluid that remains aids in the recovery of the patient, because RBCs and proteins eventually will be reabsorbed. Autotransfusion can be performed to salvage blood and reinfuse it into the anemic patient. In cases of neoplastic or traumatic uncontrollable hemorrhagic effusions, surgical exploration of the thorax is warranted.

DIAPHRAGMATIC HERNIA

Diaphragmatic hernia, or a rent in the diaphragm, can result in the protrusion of abdominal organs into the thoracic cavity and impair pulmonary expansion. Organs that are commonly herniated into the thorax include the liver, stomach, and small intestines. Diaphragmatic hernia usually is secondary to trauma but can occur as a congenital anomaly. In cases of trauma, rib fractures, pulmonary contusions, traumatic myocarditis, hemothorax, and shock are also often present concurrently with diaphragmatic hernia. Respiratory distress can be caused by any one or a combination of the above lesions. Animals with prior or chronic diaphragmatic hernias may have minimal clinical signs despite the presence of abdominal organs within the thorax. Clinical signs of acute or severe diaphragmatic hernia include respiratory distress, cyanosis, and shock.

A diagnosis of diaphragmatic hernia is made based on the patient's history (traumatic event), clinical signs, and radiographs. In some cases, ultrasonography or contrast peritoneography is necessary to confirm the diagnosis. Contrast radiographs may show the presence of the stomach or intestines within the thorax following oral administration of barium. Never administer barium directly into the peritoneal cavity or in cases of suspected gastrointestinal rupture.

Treatment of a patient with a diaphragmatic hernia includes cardiovascular and respiratory system stabilization before attempting surgical repair of the diaphragm. If the stomach is within the thorax, or if the patient's respiratory distress cannot be alleviated with medical management alone, immediate surgery is necessary. If the respiratory distress is minimal and the stomach is not located within the thorax, surgery can be postponed until the patient is a more stable anesthetic candidate. At the time of surgery, the abdominal organs are replaced into the abdominal cavity, and the rent in the diaphragm is closed. Air must be evacuated from the thorax following closure of the diaphragm. If chronic diaphragmatic hernia is repaired, the complication of reexpansion pulmonary edema can occur.

CARDIAC CHANGES ASSOCIATED WITH THORACIC TRAUMA

Cardiac injury is a common complication secondary to blunt thoracic trauma. In most cases, cardiac injury is manifested as arrhythmias, including multiple premature ventricular contractions, ventricular tachycardia, ST segment depression or elevation secondary to myocardial hypoxemia, and atrial fibrillation (See section on Cardiac Emergencies). Myocardial infarction and cardiac failure can occur. Careful and repeated assessments of the patient's blood pressure and ECG tracing should be a part of any diagnostic work-up for a patient that has sustained blunt thoracic trauma.

RIB FRACTURES AND FLAIL CHEST

Rib fractures are associated with localized pain and painful respiratory movements. Radiographs are helpful to confirm the diagnosis. Careful palpation may reveal crepitus and instability of the fractured ribs. Common problems associated with rib fractures

include pulmonary contusions, pericardial laceration, traumatic myocarditis, diaphragmatic hernia, and splenic laceration or rupture.

A flail segment results from rib fractures of more than three adjacent ribs that produce a “floating segment” of the chest wall. The flail segment moves paradoxically with respiration—that is, it moves inward during inhalation and outward during exhalation. Respiratory distress is associated with the pain caused by the fractures and the presence of traumatic underlying pulmonary pathology.

Therapy for rib fractures and flail chest includes administration of supplemental oxygen, treatment of pneumothorax or diaphragmatic hernia, and administration of systemic and local anesthesia to alleviate the discomfort associated with the fractures. Although controversial, positioning the patient with the flail segment up may reduce pain and improve ventilation. Avoid the use of chest wraps, which do nothing to stabilize the flail segment and can further impair respiratory excursions. Following administration of a systemic analgesic, administer a local anesthetic at the dorsocaudal and ventrocaudal segment of each fractured rib, and in one rib in front of and behind the flail segment. Often, pulmonary function will improve once the pain associated with rib fractures has been adequately treated. In rare cases in which the flail segment involves five or more ribs, surgical stabilization may be necessary. Single rib fractures or smaller flail segments are allowed to heal on their own.

PULMONARY DISEASES

FELINE BRONCHITIS (FELINE LOWER AIRWAY DISEASE, ASTHMA)

Feline bronchitis has a variety of names (bronchial asthma, asthma, acute bronchitis, allergic bronchitis, chronic asthmatic bronchitis, feline lower airway disease) and refers to the acute onset of respiratory distress secondary to narrowing of the bronchi. Cats may present with an acute onset of severe restrictive respiratory pattern associated with lower airway obstruction. Acute bronchitis in cats typically has an inflammatory component in the lower airways, resulting in acute bronchoconstriction, excessive mucus production, and inflammatory exudates. In cats with chronic bronchitis, there may be damage of the bronchial epithelium and fibrosis of the airways. These patients often have a history of intermittent exacerbation of clinical signs, intermittent cough, and periods of normality throughout the year. Because there appears to be an allergic or inflammatory component in feline bronchitis, clinical signs can be acutely exacerbated by stress and the presence of aerosolized particles such as perfume, smoke, and carpet powders. Causes of feline bronchitis include heartworm disease, parasitic infestation (lungworms), and (rarely) bacterial infection.

Immediate action

On presentation, the patient should be placed in an oxygen cage and allowed to rest while being observed from a distance. Postpone performing stressful diagnostic procedures until the patient's respiratory status has been stabilized. After careful thoracic auscultation, administer a short-acting bronchodilator (terbutaline, 0.01 mg/kg SQ or IM) along with a glucocorticosteroid (dexamethasone sodium phosphate 1 mg/kg IM, SQ, IV) to alleviate immediate bronchospasm and airway inflammation.

Diagnosis

Clinical signs of feline bronchitis are characterized by a short, rapid respiratory pattern with prolonged expiration with an abdominal push. Wheezes may be heard on thoracic auscultation. In some cases, no abnormalities are found on auscultation, but become acutely worse when the patient is stimulated to cough by tracheal palpation. Radiographs may reveal a hyperinflated lung field with bronchial markings and caudal displacement of the diaphragm. In some cases, consolidation of the right middle lung lobe is present. A complete blood count and serum biochemistry profile can be performed, but results usually are unrewarding. In endemic areas, a heartworm test is warranted. Fecal examination

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by flotation and the Baermann technique is helpful in ruling out lungworms and other parasites. Bronchoalveolar lavage or transtracheal wash is useful for cytologic and bacterial examination.

Management

Long-term management of feline bronchitis includes isolation from environmental exposure to potential allergens (litter dust, perfumes, smoke, incense, carpet powders) and treatment of bronchoconstriction and inflammation with a combination of oral and inhaled glucocorticosteroids and bronchodilators (Table 1-53). Antibiotic therapy is contraindicated unless a pure culture of a pathogen is documented. Oral therapy with steroids and bronchodilators should be used for a minimum of 4 weeks after an acute exacerbation and then gradually decreased to the lowest dose possible to alleviate clinical signs. Metered dose inhalers are now available (aerokat.com) for administration of inhaled bronchodilators and steroids. Fluticasone (Flovent, 100 mcg/puff) can be administered initially every 12 hours for 1 week and then decreased to once daily, in most cases. Inhaled glucocorticosteroids are not absorbed systemically, and therefore patients do not develop the adverse side effects sometimes documented with oral glucocorticosteroid administration. Because it takes time for glucocorticosteroids to reach peak effects in the lungs, administration of inhaled glucocorticosteroids should overlap with oral prednisolone administration for 5 to 7 days.

PULMONARY CONTUSIONS

Pulmonary contusions are a common sequela of blunt traumatic injury. A contusion basically is a bruise characterized by edema, hemorrhage, and vascular injury. Contusions may be present at the time of presentation or can develop over the first 24 hours after injury. A diagnosis of pulmonary contusion can be made based on auscultation of pulmonary crackles, presence of respiratory distress, and the presence of patchy interstitial to alveolar infiltrates on thoracic radiographs. Radiographic signs can lag behind the development of clinical signs of respiratory distress and hypoxemia by 24 hours.

TABLE 1-53 Drugs to Use in the Immediate and Long-Term Management of Feline Bronchitis

Drug	Emergency treatment	Long-term management
Bronchodilators		
Aminophylline	4 mg/kg IM (emergency)	5 mg/kg PO q8-12h
Terbutaline	0.01 mg/kg SQ	0.625 mg/cat PO q12h
Theophylline		50-100 mg/cat PO q24h
Albuterol MDI*	90 µg	90 µg as needed up to q6h
Glucocorticosteroids		
Dexamethasone sodium phosphate	1 mg/kg IV, IM, SQ	
Dexamethasone		0.25 mg/kg PO q8-12h, then taper to q24h for 1-2 months
Prednisolone		1 mg/kg PO q12h, then taper
Prednisolone sodium succinate	50-100 mg/cat IV	0.1-0.625 mg/kg PO q12h
Triamcinolone	0.11 mg/kg SQ, repeat	0.11 mg/kg PO q12-24h, then taper in 10-14 days
Fluticasone MDI	110 µg/puff	110 µg MDI q12h
Beclomethasone	220 µg/puff	220 µg MDI q6-8h

*MDI, Metered dose inhaler.

Treatment of pulmonary contusions is supportive. Administer supplemental oxygen in a manner that is least stressful for the animal. Arterial blood gas analysis or pulse oximetry can determine the degree of hypoxemia and monitor the response to therapy. Intravenous fluids should be administered with caution to avoid exacerbating pulmonary hemorrhage or fluid accumulation in the alveoli. Treat other conditions associated with the traumatic event. Possible complications of pulmonary contusions are rare but include bacterial infection, abscessation, lung lobe consolidation, and the development of cavitary lesions. The routine use of antibiotics or steroids in cases of pulmonary contusions is contraindicated unless external wounds are present. Empiric antibiotic use without evidence of external injury or known infection can potentially increase the risk of a resistant bacterial infection. Steroids have been shown to decrease pulmonary alveolar macrophage function and impair wound healing and are contraindicated.

ASPIRATION PNEUMONIA

Aspiration pneumonia can occur in animals as a result of abnormal laryngeal or pharyngeal protective mechanisms or can be secondary to vomiting during states of altered mentation, including anesthesia, recovery from anesthesia, and sleep. Megaesophagus, systemic polyneuropathy, myasthenia gravis, and localized oropharyngeal defects such as cleft palate can increase the risk of developing aspiration pneumonitis. Iatrogenic causes of aspiration pneumonia include improper placement of nasogastric feeding tubes, overly aggressive force-feeding, and oral administration of drugs. Aspiration of contents into the airways can cause mechanical airway obstruction, bronchoconstriction, chemical damage to the alveoli, and infection. Severe inflammation and airway edema are common. Pulmonary hemorrhage and necrosis can occur.

Diagnosis of aspiration pneumonia is based on clinical signs of pulmonary parenchymal disease, a history consistent with vomiting or other predisposing causes, and thoracic radiographs demonstrating a bronchointerstitial to alveolar pulmonary infiltrate. The most common site is the right middle lung lobe, although the pneumonia can occur anywhere, depending on the position of the patient at the time of aspiration. A transtracheal wash or bronchoalveolar lavage is useful for bacterial culture and susceptibility testing.

Treatment of aspiration pneumonia includes antibiotic therapy for the infection, administration of supplemental oxygen, and loosening the debris in the airways. Administer intravenous fluids to maintain hydration. Nebulization with sterile saline and chest physiotherapy (coupage) should be performed at least every 8 hours. Antibiotics to consider in the treatment of aspiration pneumonia include ampicillin/enrofloxacin, amoxicillin-clavulanate, ampicillin-sulbactam, trimethoprim sulfa, and chloramphenicol. The use of glucocorticosteroids is absolutely contraindicated. Continue antibiotic therapy for a minimum of 2 weeks after the resolution of radiographic signs of pneumonia.

PULMONARY EDEMA

Pulmonary edema arises from the accumulation of fluid in the pulmonary interstitial alveolar spaces, and airways. Ventilation-perfusion abnormalities result in hypoxia. Pulmonary edema can be caused by increased pulmonary vasculature hydrostatic pressure, decreased pulmonary oncotic pressure, obstruction of lymphatic drainage, or increased capillary permeability. Multiple factors can occur simultaneously. The most common cause of edema is increased pulmonary hydrostatic pressure resulting from left-sided congestive heart failure. Decreased plasma oncotic pressure with albumin <1.5 g/dL can also result in accumulation of fluid in the pulmonary parenchyma. Overzealous intravenous crystalloid fluid administration can result in dilution of serum oncotic pressure and vascular overload. Obstruction of lymphatic drainage is usually caused by neoplasia. Other causes of pulmonary edema include pulmonary thromboembolic disease, severe upper airway obstruction (noncardiogenic pulmonary edema), seizures, and head trauma.

Increased capillary permeability is associated with a variety of diseases that cause severe inflammation (systemic inflammatory response syndrome). The resultant pulmonary edema contains a high amount of protein and is known as acute respiratory

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distress syndrome (ARDS). ARDS can be associated with pulmonary or extrapulmonary causes, including direct lung injury from trauma, aspiration pneumonia, sepsis, pancreatitis, smoke inhalation, oxygen toxicity, electrocution, and immune-mediated hemolytic anemia with disseminated intravascular coagulation.

Diagnosis of pulmonary edema is made based on clinical signs of respiratory distress and the presence of crackles on thoracic auscultation. In severe cases, cyanosis and fulminant blood-tinged frothy edema fluid may be present in the mouth and nostrils. Immediate management includes administration of furosemide (4-8 mg/kg IV, IM) and supplemental oxygen. Sedation with low-dose morphine sulfate (0.025-0.1 mg/kg IV) is helpful in dilating the splanchnic capacitance vasculature and relieving anxiety for the patient. If fluid overload is suspected secondary to intravenous fluid administration, fluids should be discontinued. Severely hypoalbuminemic patients should receive concentrated human albumin (2 mL/kg of a 25% solution) or fresh frozen plasma. Furosemide as a constant rate infusion (0.66-0.1 mg/kg/hour) also can dilate the pulmonary vasculature and decrease fluid accumulation in cases of ARDS. Following initial stabilization of the patient, thoracic radiographs and an echocardiogram should be assessed to determine cardiac side, pulmonary vascular size, and cardiac contractility. Further diagnostic testing may be required to determine other underlying causes of pulmonary edema.

Heart failure is managed with vasodilators, diuretics, oxygen, and sometimes positive inotropes. Treatment ultimately consists of administration of supplemental oxygen, minimal stress and patient handling, and judicious use of diuretics. In cases of cardiogenic pulmonary edema, administer furosemide (4-8 mg/kg IV, IM) every 30 to 60 minutes until the patient loses 7% of its body weight. Positive inotropic and antiarrhythmic therapy may be necessary to improve cardiac contractility and control dysrhythmias. The clinician should determine whether the cause of the pulmonary edema is secondary to congestive heart failure with pulmonary vascular overload, volume overload, hypoalbuminemia, or increased permeability (ARDS). Pulmonary edema secondary to ARDS typically is refractory to supplemental oxygen and diuretic therapy. In many cases, mechanical ventilation should be considered.

PULMONARY THROMBOEMBOLISM

A diagnosis of pulmonary thromboembolism (PTE) is difficult to make and is based on clinical signs of respiratory distress consistent with PTE, lack of other causes of hypoxemia, a high index of suspicion in susceptible animals, the presence of a condition associated with PTE, and radiographic findings. Virchow's triad consists of vascular endothelial injury, sluggish blood flow with increased vascular stasis, and a hypercoagulable state as predisposing factors for thromboembolic disease. Clinical conditions that predispose an animal to PTE include hyperadrenocorticism, disseminated intravascular coagulation (DIC), catheterization of blood vessels, bacterial endocarditis, protein-losing nephropathy or enteropathy, hyperviscosity syndromes, heat-induced illness, pancreatitis, diabetes mellitus, inflammatory bowel disease, and immune-mediated hemolytic anemia. Definitive diagnosis requires angiography or a lung perfusion scan.

Clinical signs associated with PTE include an acute onset of tachypnea, tachycardia, orthopnea, and cyanosis. If the embolism is large, the patient may respond poorly to supplemental oxygen administration. Pulmonary hypertension can cause a split second heart sound on cardiac auscultation. In some cases, a normal thoracic radiograph is present in the face of severe respiratory distress. This is a classic finding in cases of PTE. Potential radiographic abnormalities include dilated, tortuous, or blunted pulmonary arteries; wedge-shaped opacities in the lungs distal to an obstructed artery; and interstitial to alveolar infiltrates. The right heart may be enlarged.

Echocardiography can show right heart enlargement, tricuspid regurgitation, pulmonary hypertension, and evidence of underlying cardiac disease, possibly with clots in the atria. Measurement of antithrombin (AT) and D-dimer levels can be useful in the identification of hypercoagulable states, including DIC. Treatment of any patient with AT deficiency or DIC includes replenishment of AT and clotting factors in the form of fresh frozen plasma.

Treatment of PTE includes therapy for cardiovascular shock, oxygen supplementation, and thrombolytic therapy (see section on Thromboembolic Therapy). For short-term treatment, administer heparin (heparin sodium, 200-300 units/kg SQ once, followed by 100 units/kg q8h of unfractionated heparin; or fractionated heparin). Thrombolytic therapy may include tissue plasminogen activator, streptokinase, or urokinase. Long-term therapy with low molecular weight heparin or warfarin may be required to prevent further thromboembolic events. Ideally, management should include treatment and elimination of the underlying disease.

SMOKE INHALATION

Smoke inhalation commonly occurs when an animal is trapped in a burning building. The most severe respiratory complications of smoke inhalation are seen in animals that are close enough to the flames to also sustain burn injuries (see section on Burn Injury). At the scene, many animals are unconscious from the effects of hypoxia, hypercapnia, carbon monoxide intoxication, and hydrogen cyanide gases that accumulate in a fire. Carbon monoxide produces hypoxia by avidly binding to and displacing oxygen binding to hemoglobin, resulting in severe impairment of oxygen-carrying capacity. The percentage of carboxyhemoglobin in peripheral blood depends on the amount of carbon monoxide in inhaled gases and the length of time of exposure. Clinical signs of carbon monoxide intoxication include cyanosis, nausea, vomiting, collapse, respiratory failure, loss of consciousness, and death.

Smoke inhalation of superheated particles also causes damage to the upper airways and respiratory tree. The larynx can become severely edematous and obstruct inspiration. Emergency endotracheal intubation, tracheal oxygen, or tracheostomy tube may be required in the initial resuscitation of the patient, depending on the extent of airway edema. Inhalation of noxious gases and particles can cause damage to the terminal respiratory bronchioles. Specific noxious gases that can cause alveolar damage include combustible particles from plastic, rubber, and other synthetic products. Pulmonary edema, bacterial infection, and ARDS can result.

In any case of smoke inhalation, the first and foremost treatment is to get the animal away from the source of the flames and smoke and administer supplemental oxygen at the scene. At the time of presentation, carefully examine the animal's eyes, mouth, and oropharynx. Suction soot and debris from the mouth and upper airways. Evaluate the patient's respiratory rate, rhythm, and pulmonary sounds. Arterial blood gases should be analyzed with co-oximetry to evaluate the PaO₂ and carboxyhemoglobin concentrations. Evaluation of SaO₂ by pulse oximetry is not accurate in cases of smoke inhalation, as the PaO₂ may appear normal, even when large quantities of carboxyhemoglobin are present. Radiographs are helpful in determining the extent of pulmonary involvement, although radiographic signs may lag behind the appearance of clinical respiratory abnormalities by 16 to 24 hours. Bronchoscopy and bronchoalveolar lavage provide a more thorough and accurate evaluation of the respiratory tree; however, these procedures should be performed only in patients whose cardiovascular and respiratory status is stable.

Management of the patient with smoke inhalation includes maintaining a patent airway, administration of supplemental oxygen, correction of hypoxemia and acid-base abnormalities, preventing infection, and treating thermal burns (See section on Burn Injury). If severe laryngeal edema is present, a temporary tracheostomy may be necessary to allow adequate oxygenation and ventilation. Glucocorticosteroids should NOT be empirically used in the treatment of smoke inhalation, because of the risk of decreasing pulmonary alveolar macrophage function and increasing the potential for infection. In cases of severe laryngeal edema, however, glucocorticosteroids may be necessary to decrease edema and inflammation. The use of empiric antibiotics is contraindicated unless clinical signs of deterioration and bacterial pneumonia develop.

EPISTAXIS

Epistaxis can be caused by facial trauma, a foreign body, bacterial or fungal rhinitis, neoplasia, coagulopathies, and systemic hypertension. Acute, severe bilateral hemorrhage without

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exudate is suggestive of a systemic disorder. A history of chronic nasal discharge usually accompanies nasal disease. Acute unilateral epistaxis can occur with nasal or systemic disease.

In most cases, cage rest is sufficient to temporarily diminish blood loss. Sedation (acepromazine, 0.02-0.05 mg/kg IV, IM, SQ) may be helpful in alleviating anxiety and decreasing blood pressure. The hypotensive effects of acepromazine are potentially harmful if severe blood loss has occurred. If evidence of hypovolemia is present (see section on Hypovolemic Shock), intravenous fluid resuscitation should be administered.

Rapid assessment of clotting ability, with a platelet count estimate and clotting profile (ACT or APTT and PT), should be performed. If epistaxis secondary to Vitamin K antagonist rodenticide intoxication is suspected, administer vitamin K₁ and fresh frozen plasma or fresh whole blood.

Persistent hemorrhage from a nasal disorder can be treated with dilute epinephrine (1:100,000) into the nasal cavity with the nose pointed toward the ceiling to promote vasoconstriction. If this fails, the animal can be anesthetized, and the nasal cavity packed with gauze, and the caudal oropharynx and external nares covered with umbilical tape to control hemorrhage. A rhinoscopy should be performed to determine the cause of ongoing hemorrhage. Continued excessive hemorrhage can be controlled with ligation of the carotid artery on the side of the hemorrhage, or with percutaneous arterial embolization.

Additional Reading

- Buerge HCD: Pleural effusion in cats. *Vet Med* 97(11):812-818, 2002.
- Campbell VL, King LG: Pulmonary function, ventilator management, and outcome of dogs with thoracic trauma and pulmonary contusions: 10 cases (1994-1998). *J Am Vet Med Assoc* 217(10):1505-1509, 2000.
- Carpenter DH, Macintire DK, Tyler JW: Acute lung injury and acute respiratory distress syndrome. *Comp Cont Educ Pract Vet* 23(8):712-725, 2001.
- Drobatz KJ, Walker LM, Hendricks JC: Smoke exposure in cats: 22 cases (1986-1997). *J Am Vet Med Assoc* 215(9):1312-1316, 1999.
- Drobatz KJ, Walker LM, Hendricks JC: Smoke exposure in dogs: 27 cases (1988-1997). *J Am Vet Med Assoc* 215(9):1306-1311, 1999.
- Fossum TW, Mertens MM, Miller MW, et al: Thoracic duct ligation and pericardectomy for treatment of idiopathic chylothorax. *J Vet Intern Med* 18(3):307-310, 2004.
- Gellasch KL, De Costa Gomez T, McAnulty JE, Bjorling DE, et al. Use of intraluminal nitinol stents in the treatment of tracheal collapse in a dog. *J Am Vet Med Assoc* 221(12):1719-1723, 2002.
- Gieger T, Northrup N: Clinical approach to epistaxis. *Comp Cont Educ Pract Vet* 26(1):30-43, 2004.
- Hughes D: Pulmonary edema. In Wingfield WE, Raffé MR (eds): *The Veterinary ICU Book*. Teton NewMedia, Jackson, Wyo, 2001.
- Hyun C: Radiographic diagnosis of diaphragmatic hernia: review of 60 cases in dogs and cats. *J Vet Sci* 5(2):157-162, 2004.
- Johnson L: Tracheal collapse: diagnosis and medical and surgical management. *Vet Clin North Am Small Anim Pract* 30(6):1253-1266, 2000.
- King LG, Waddell LS: Acute respiratory distress syndrome. In Wingfield WE, Raffé MR (eds): *The Veterinary ICU Book*. Teton NewMedia, Jackson, Wyo, 2001.
- Koch DA, Arnold S, Hubler M, Montavon PM: Brachycephalic syndrome in dogs. *Comp Cont Educ Pract Vet* 25(1):48-55, 2003.
- MacPhail CM, Monnet E: Outcome and postoperative complications in dogs undergoing surgical treatment of laryngeal paralysis: 140 cases (1985-1998). *J Am Vet Med Assoc* 218(12):1949-1956, 2001.
- Mariani CL: Full recovery following delayed neurologic signs after smoke inhalation in a dog. *J Vet Emerg Crit Care* 13(4):235-239, 2003.
- Mazzaferro EM: Aspiration pneumonitis. In Wingfield WE, Raffé MR (eds): *The Veterinary ICU Book*. Teton NewMedia, Jackson, Wyo, 2001.
- Mazzaferro EM: Respiratory Injury. In Wingfield WE, Raffé MR (eds): *The Veterinary ICU Book*. Teton NewMedia, Jackson, Wyo, 2001.
- McKiernan BC, Miller C: Allergic airway disease. In Wingfield WE, Raffé MR (eds): *The Veterinary ICU Book*. Teton NewMedia, Jackson, Wyo, 2001.

- Mellanby RJ, Villiers E, Herrtage ME: Canine pleural and mediastinal effusion, a retrospective study of 81 cases. *J Small Anim Pract* 43(10):447-451, 2002.
- Mueller ER: Suggested strategies for ventilatory management in veterinary patients with acute respiratory distress syndrome. *J Vet Emerg Crit Care* 11(3):191-197, 2001.
- Reiss AJ, McKiernan BC: Laryngeal and tracheal disorders. In Wingfield WE, Raffae MR, editors: *The veterinary ICU book*, Jackson, Wyo, 2001, Teton NewMedia,.
- Reiss AJ, McKiernan BC. Pneumonia. In Wingfield WE, Raffae MR (eds): *The Veterinary ICU Book*. Teton NewMedia, Jackson, Wyo, 2001.
- Rooney MB, Monnet E: Medical and surgical treatment of pyothorax in dogs: 26 cases (1991-2001). *J Am Vet Med Assoc* 221(1):86-92, 2002.
- Schmidt CW, Tobias KM, McCrackin Stevenson MA: Traumatic diaphragmatic hernia in cats: 34 cases (1991-2000). *J Am Vet Med Assoc* 229(9):1237-1240, 2003.
- Scott JA, Macintire DK: Canine Pyothorax: Clinical presentation, diagnosis, and treatment. *Comp Cont Educ Pract Vet* 25(3):180-194, 2003.
- Scott JA, Macintire DK: Canine pyothorax: pleural anatomy and pathophysiology. *Comp Cont Educ Pract Vet* 25(3):172-179, 2003.
- Smeak DD, Birchard SJ, McLoughlin MA, et al: Treatment of chronic pleural effusion with pleuroperitoneal shunt in dogs: 14 cases (1985-1999). *J Am Vet Med Assoc* 219(11):1590-1597, 2001.
- Tobias KM, Jackson AM, Harvey RC: Effects of doxapram hydrochloride on laryngeal function of normal dogs and dogs with naturally occurring laryngeal paralysis. *Vet Anaesth Analg* 31(4):258-263, 2004.
- Vassilev E, McMichael M: An overview of positive pressure ventilation. *J Vet Emerg Crit Care* 14(1):15-21, 2004.
- Waddell LS, Brady CA, Drobatz KJ: Risk factors, prognostic indicators, and outcome of pyothorax in cats: 80 cases (1986-1999). *J Am Vet Med Assoc* 221(6):819-824, 2002.
- Weiss C, Nicholson ME, Rollings C, et al: Use of percutaneous arterial embolization for the treatment of intractable epistaxis in 3 dogs. *J Am Vet Med Assoc* 224(8):1307-1311, 2004.

SUPERFICIAL SOFT TISSUE INJURIES

Wounds have been classified in several ways according their degree of tissue integrity, etiologic force, degree of contamination and duration, and degree of contamination and infection (Table 1-54). There are also unique causes of wounds such as burns, psychogenic dermatoses, frostbite, decubital ulcers, and snake bite.

The animal should be transported to the nearest veterinary facility for definitive care. The wound should be covered or packed with dry gauze or clean linen to protect the wound, and to prevent further hemorrhage and contamination. If an open fracture is present, the limb should be splinted without placing the exposed bone back into the wound. Replacing the exposed bone fragment back through the skin wound can cause further damage to underlying soft tissue structures and increase the degree of contamination of deeper tissues. If a spinal fracture is suspected, the patient should be transported on a stable flat surface to prevent further spinal mobilization and neurologic injury.

At the time of presentation, first refer to the ABCs of trauma, taking care to evaluate and stabilize the patient's cardiovascular and respiratory status. After a complete physical examination and history, ancillary diagnostic techniques can be performed if the patient is hemodynamically stable (see section on Triage, Assessment, and Treatment of Emergencies).

WOUND MANAGEMENT

Initially, every patient with superficial wound should receive some degree of analgesia and an injection of a first-generation cephalosporin, preferably within 3 hours of the injury. Evaluate the wound after the patient's cardiovascular and respiratory status have been stabilized. Always cover an open wound before taking an animal to the hospital to prevent a nosocomial infection. Evaluate limb wounds for neural, vascular, and orthopedic abnormalities. Carefully examine the structures deep to the superficial wounds.

When there has been a delay in assessment of the wound, obtain samples for culture and antimicrobial susceptibility testing. If the wound is older and obviously infected, a Gram stain can help guide appropriate antimicrobial therapy pending results of culture

TABLE 1-54 Classification of Soft Tissue Wounds

Classification	Characteristics
<i>Tissue integrity</i>	
Open	Lacerations or skin loss
Closed	Crushing injuries and contusions
<i>Etiologic force</i>	
Abrasion	Loss of epidermis and portions of dermis, usually caused by shearing between two compressive surfaces
Avulsion	Tearing of tissue from its attachment because of forces similar to those causing abrasion but of a greater magnitude
Incision	Wound created by a sharp object; wound edges are smooth and there is minimal trauma in the surrounding tissues
Laceration	Irregular wound caused by tearing of tissue with variable damage to the superficial and underlying tissue
Puncture	Penetrating wound caused by a missile or sharp object; superficial damage may be minimal; damage to deeper structures may be considerable; contamination by fur and bacteria with subsequent infection is common
<i>Degree of contamination and duration</i>	
Class I	0-6 hours with minimal contamination
Class II	6-12 hours with significant contamination
Class III	>12 hours with gross contamination
<i>Degree of contamination or infection</i>	
Clean wound	Surgically created under aseptic conditions; no invasion of the respiratory, gastrointestinal, or genitourinary tracts, or of the oropharyngeal cavity
Clean contaminated wound	Minimal contamination, and contamination can be removed effectively; includes operative wounds involving the respiratory, gastrointestinal, and genitourinary tracts
Contaminated wound	Open traumatic wound with heavy contamination and possibly foreign debris; includes operative wounds with major breaks in aseptic technique and incisions in areas of acute nonpurulent inflammation adjacent to inflamed or contaminated skin
Dirty/infected wound	Old traumatic wound and wounds with clinical signs of infection or perforated viscera

Modified from Swaim SF, Henderson RA: *Small animal wound management*, ed 2, Media, Pa, 1997, Williams & Wilkins.

and susceptibility testing. Place a support bandage saturated with a water-soluble antibiotic ointment or nonirritating antimicrobial solution (e.g., 0.05% chlorhexidine, if bone or joint tissue is not exposed) around the wound. In addition to a first-generation cephalosporin, other appropriate antibiotic choices include amoxicillin-clavulanate, trimethoprim-sulfadiazine, amoxicillin, and ampicillin. If gram-negative flora are present, administer enrofloxacin. Administer the antibiotics of choice for a minimum of 7 days unless a change of antibiotic therapy is indicated.

At the time of wound cleansing or definitive wound repair, the patient should be placed under general anesthesia with endotracheal intubation, unless the procedure will be brief (i.e., less than 10 minutes). In such cases, a short-acting anesthetic combination

(analgesia + propofol, analgesia + ketamine/diazepam) can be administered to effect. Heavy sedation with infiltration of a local anesthetic may also be appropriate for very small wounds, depending on the location of the wound and temperament of the patient. Protect the wound by packing it with sterile gauze sponges soaked in sterile saline, or with water-soluble lubricating gel such as K-Y jelly.

Clip the fur surrounding the wound, moving from the inner edge of the wound outward, to help prevent wound contamination with fur or other debris. Scrub the wound and surrounding skin with an antimicrobial soap and solution such as dilute chlorhexidine until the area is free of all gross debris. Gross debris within the wound itself can be flushed using a 30-mL syringe filled with sterile saline or lactated Ringer's solution and an 18-gauge needle. Pressure-lavage systems are also available for use, if desired. Grossly contaminated wounds can be rinsed first with warm tap water to eliminate gross contamination, and then prepared as just described.

Debride the wound, removing skin and other soft tissue that is not obviously viable. Obviously viable and questionable tissue should remain, and the wound left open for frequent reassessment on a daily basis. Remove any dark or white segments of skin. Questionable skin edges may or not regain viability and should be left in place for 48 hours, so the wound can fully reveal itself. Excise grossly contaminated areas of fat and underlying fascia. Blood vessels that are actively bleeding should be ligated to control hemorrhage, if collateral circulation is present.

If nerve bundles are ligated cleanly in a clean wound, the nerve edges should be reapposed and anastomosed. If gross contamination is present, however, definitive neurologic repair should be delayed until healthy tissue is present. Excise contaminated muscle until healthy bleeding tissue is present. Anastomose tendon lacerations if the wound is clean and not grossly contaminated. If gross contamination is present, the tendon can be temporarily anastomosed and a splint placed on the limb until definitive repair of healthy tissue is possible.

Thoroughly lavage open wounds to a joint with sterile saline or lactated Ringer's solution. Infusion of chlorhexidine or povidone-iodine solution into the joint can cause a decrease in cartilage repair and is contraindicated. Smooth sharp edges and remove any obvious fragments. Whenever possible, the joint capsule and ligaments should be partially or completely closed. After removing bullets and metal fragments, the subcutaneous tissue and skin should be left open to heal by second intention, or should be partially closed with a drain. The joint should then be immobilized.

Injuries and exposed bone should be carefully lavaged, taking care to remove any gross debris without pushing the debris further into the bone and wound. The bone should be covered with a moist dressing and stabilized until definitive fracture repair can be made. This type of injury typically is seen with shearing injuries of the distal extremities caused by interaction with slow-moving vehicles. Perform wet-to-dry or enzymatic debridement until a healthy granulation bed is present.

If large areas of contamination are present (e.g., necrotizing fasciitis), en bloc debridement may be necessary. En bloc debridement consists of complete excision of badly infected wounds without entering the wound cavity, to prevent systemic infection. This technique should be used only if there is sufficient skin and soft tissue to allow later closure and it can be performed without damaging any major nerves, tendons, or blood vessels.

OPEN WOUNDS

Open wounds often are managed by second intention healing, delayed primary closure, or secondary closure. See section on Wound Management and Bandaging for a more complete discussion on the use of various bandaging materials in the treatment of open wounds.

CLOSED WOUNDS

If an animal is presented very shortly after a wound has occurred and there is minimal contamination and trauma, the wound can be closed after induction of anesthesia and

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careful preparation of the wound and surrounding tissues. Close any dead space under the skin with absorbable suture material in an interrupted suture pattern. Avoid incising major blood vessels or nerves. Close the subcutaneous tissues with absorbable suture material in an interrupted or continuous suture pattern. Take care that there is not too much tension on the wound, or else surgical dehiscence will occur with patient movement. Close the skin with nonabsorbable suture or surgical staples (2-0 to 4-0).

If there is any doubt at the time of repair about tissue status or inability to close all dead space, place a passive drain (Penrose drain) so that the proximal end of the drain is anchored in the proximal aspect of the wound with a suture(s). Leave the ends long so that the suture can be accurately identified at the time of drain removal. Pass the suture through the skin, through the drain, and out the other side of the skin. Place the rest of the drain into the wound and then secure it at the most ventral portion of the wound or exit hole in the most dependent area of the body, to allow drainage and prevent seroma formation. Close the subcutaneous tissue over the drain before skin closure. During wound closure, be sure to not incorporate the subcutaneous or skin sutures into the drain, or it will not be possible to remove the drain without reopening the wound. Bandage the area to prevent contamination. The drain can be removed once drainage is minimal (usually 3 to 5 days).

Active drains can be constructed or purchased; their use is indicated in wounds that are free of material that can plug the drain. To construct a small suction drain, remove the female portion or catheter hub at the end of a butterfly catheter. Fenestrate the tubing so that there are multiple side holes, taking care to avoid making the holes larger than 50% of the circumference of the tubing. Place the tubing into the wound via a small stab incision distal to the wound. Use a purse-string suture around the tubing to facilitate a tight seal and prevent the tubing from exiting the wound. Following wound closure, insert the butterfly needle into a 5- to 10-mL evacuated blood collection tube to allow fluid to drain into the tube. Incorporate the tube into the bandage, and replace it when it becomes full.

Alternatively, the butterfly portion of the system can be removed and the tube fenestrated as described previously. Place the tube into the wound and suture it in place to create a tight seal. Secure the catheter hub to a syringe in which the plunger has been drawn back slightly to create suction. Insert a metal pin or 16- to 18-gauge needle through the plunger at the top of the barrel to hold it at the desired level. Incorporate the suction apparatus into the bandage and replace it when it becomes full.

DELAYED PRIMARY CLOSURE

Delayed primary closure should be considered when there is heavy contamination, purulent exudate, residual necrotic debris, skin tension, edema and erythema, and lymphangitis. Delayed primary closure usually is made 3 to 5 days after the initial wound infliction and open wound management has been performed. Once healthy tissue is observed, the skin edges should be debrided and the wound closed as with primary closure.

SECONDARY WOUND CLOSURE

Secondary wound closure should be considered when infection and tissue trauma necessitate open wound management for more than 5 days. Secondary wound closure is performed after the development of a healthy granulation bed. This technique also is useful when a wound has dehisced and has formed granulation tissue.

If the wound edges can be manipulated into apposition and if epithelialization has not begun, the wound can be cleansed and the wound edges apposed and sutured. This is known as early secondary closure.

Late secondary closure should be performed whenever there is a considerable amount of granulation tissue, the edges of the wound cannot be manipulated into position, and epithelialization has already started. In such cases, the wound should be cleaned, and the skin edges debrided to remove the epithelium. The remaining wound edges are then sutured over the granulation tissue (Table 1-55).

TABLE 1 - 55 Complicating Factors Involving the Management of Superficial Soft Tissue Wounds

Circumstance	Potential problem(s)
Improper handling of animal during transport	Further tissue and neurologic damage may occur (e.g., improper limb or spine immobilization).
Inadequate assessment of animal's general condition or wounded tissues	Animal's condition may worsen or animal may succumb; tissue injuries may be overlooked.
Inadequate wound protection during assessment, resuscitation, or stabilization procedures	Further wound contamination may occur at veterinary facility.
Inadequate wound protection while preparing the surrounding area	Further wound contamination with fur and debris may occur.
Insufficient wound lavage	Wound infection may occur.
Hydrogen peroxide wound lavage	Lavage offers little bactericidal activity and contributes to irritation of tissues and delayed healing.
Povidone-iodine wound lavage	Lavage has short residual activity and absorption with large wound.
Overly aggressive initial layered debridement	Debridement may result in the removal of viable tissue.
En bloc debridement	Debridement results in removal of large amounts of tissue and a large defect for closure.
Use of drains	Potential exists for bacteria to ascend along the drain, for drain removal by the animal or breakage of the drain, and for possible tissue emphysema with air being sucked under the skin with patient movement.
Tube-type drains	Drains may cause postoperative discomfort; fenestrations may become occluded to stop intraluminal drainage.
Deeply placed sutures in the presence of a drain	Drain may be incorporated into the repair and prevent drain removal.
Active drains	High negative pressure may cause tissue injury; highly productive wounds may necessitate changing the evacuated blood tubes several times a day with constructed drains.

Additional Reading

Swaim SF, Henderson RA: small animal wound management. 2nd Edition. Williams and Wilkins, Media, Pa, 1997,

SHOCK

Shock is defined as a state of inadequate circulating volume and inability to meet cellular oxygen demands. There are three types of shock: hypovolemic, cardiogenic, and septic. Early recognition of the type of shock present is crucial in the successful clinical management of shock syndrome. Tissue oxygen delivery is based on cardiac output and arterial oxygen concentration. Knowledge of the components of normal oxygen delivery is essential to the treatment of shock in the critical patient.

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Oxygen delivery (DO_2) = cardiac output (Q) \times arterial oxygen content (CaO_2)

where Q = heart rate \times stroke volume. Stroke volume is affected by preload, afterload, and cardiac contractility.

$$\text{CaO}_2 = (1.34 \times [\text{Hb}] \times \text{SaO}_2) + (0.003 \times \text{PaO}_2)$$

where Hb = hemoglobin concentration, SaO_2 = oxygen saturation, and PaO_2 = arterial partial pressure of oxygen in mm Hg.

Thus, factors that can adversely affect oxygen delivery include inadequate preload or loss of circulating volume, severe peripheral vasoconstriction and increased afterload, depressed cardiac contractility, tachycardia and decreased diastolic filling, cardiac dysrhythmias, inadequate circulating hemoglobin, and inadequate oxygen saturation of hemoglobin. During septic shock, enzymatic dysfunction and decreased cellular uptake and utilization of oxygen also contribute to anaerobic glycolysis.

An inadequate circulating volume may develop secondary to maldistribution of available blood volume (traumatic, septic, and cardiogenic origin) or as a result of absolute hypovolemia (whole blood or loss of extracellular fluid). Normally, the animal compensates by (1) splenic and vascular constriction to translocated blood from venous capacitance vessels to central arterial circulation, (2) arteriolar constriction to help maintain diastolic blood pressure and tissue perfusion, and (3) an increase in heart rate to help maintain cardiac output. Arteriolar vasoconstrictions support perfusion to the brain and heart at the expense of other visceral organs. If vasoconstriction is severe enough to interfere with delivery of adequate tissue oxygen for a sufficient period of time, the animal may die.

HYPVOLEMIC SHOCK

Hypovolemic shock can result from acute hemorrhage or from severe fluid loss from vomiting, diarrhea, or third spacing of fluids. Early in shock, baroreceptors in the carotid body and aortic arch sense a decrease in wall stretch from a decrease in circulating fluid volume. Tonic inhibition of sympathetic tone via vagal stimulation is diminished, and heart rate and contractility increase and peripheral vessels constrict to compensate for the decrease in cardiac output. The compensatory mechanisms protect and support blood supply to the brain and heart at the expense of peripheral organ perfusion. This is called *early compensatory shock*.

Early compensatory shock is characterized by tachycardia, normal to fast capillary refill time, tachypnea, and normothermia. As shock progresses, the body loses its ability to compensate for ongoing fluid losses. Early decompensatory shock is characterized by tachycardia, tachypnea, delayed capillary refill time, normotension to hypotension, and a fall in body temperature. End-stage decompensatory shock is characterized by bradycardia, markedly prolonged capillary refill time, hypothermia, and hypotension. Aggressive treatment is necessary for any hope of a favorable outcome.

SEPTIC SHOCK

Septic shock should be considered in any patient with a known infection, recent instrumentation that could potentially introduce infection (indwelling intravenous or urinary catheter, surgery or penetrating injury), disorders or medical therapy that can compromise immune function (diabetes mellitus, immunodeficiency virus, parvovirus or feline panleukopenia virus infection, stress, malnutrition, glucocorticoids, chemotherapy). The presence of bacteria, viruses or rickettsiae, protozoa, or fungal organisms in the blood constitutes septicemia. Septic shock is characterized by the presence of sepsis and refractory hypotension that is unresponsive to standard aggressive fluid therapy and inotropic or pressor support. Septic shock and other causes of inflammation can lead to systemic inflammatory response syndrome (SIRS). In animals, the presence of two or more of the criteria in Table 1-56 in the presence of suspected inflammation or sepsis constitutes SIRS (Table 1-56).

TABLE 1 - 56 Summary of Systemic Inflammatory Response Syndrome Criteria

Criteria	Dogs	Cats
Temperature	<100° F or >103.5° F	<100° F or >103.5° F
Heart rate	>120 beats/minute in dogs	<140 or >250 beats/minute in cats
Respiratory rate	>20 breaths/minute or PaCO ₂ <32 mm Hg	>40 breaths/minute or PaCO ₂ <32 mm Hg
White blood cell count	>18,000 cells/μL or <4000 cells/μL or >10% bands	19,000 cells/μL or <5000 cells/mL or >10% bands

Clinical signs associated with sepsis may be vague and nonspecific, including weakness, lethargy, vomiting, and diarrhea. Cough and pulmonary crackles may be associated with pneumonia. Decreased lung sounds may be associated with pyothorax. Abdominal pain and fluid may be associated with septic peritonitis. Vaginal discharge may or may not be present in patients with pyometra. Diagnostic tests should include a white blood cell count, serum biochemical profile, coagulation tests, thoracic and abdominal radiographs, and urinalysis.

The white blood cell count in a septic patient that is appropriately responding to the infection will be elevated with a left-shifted neutrophilia and leukocytosis. A degenerative left shift, in which leukopenia with elevated band neutrophils suggests an overwhelming infection. Biochemical analyses may demonstrate hypoglycemia and nonspecific hepatocellular and cholestatic enzyme elevations. In the most severe cases, metabolic (lactic) acidosis, coagulopathies, and end-organ failure, including anuria and ARDS, may be present.

CARDIOGENIC SHOCK

Cardiogenic shock occurs as a result of cardiac output inadequate to meet cellular oxygen demands. Cardiogenic shock is associated with primary cardiomyopathies, cardiac dysrhythmias, pericardial fluid, and pericardial fibrosis. Abnormalities seen on physical examination often are similar to those seen in other categories of shock, but they can also include cardiac murmurs, dysrhythmias, pulmonary rales, bloody frothy pulmonary edema fluid from the nares or mouth, orthopnea, and cyanosis. It is important to distinguish the primary cause of shock before implementing treatment (Table 1-57), whenever possible, because treatment for a suspected ruptured hemangiosarcoma differs markedly from the treatment for end-stage dilatative cardiomyopathy. The patient's clinical signs may be similar and include a peritoneal fluid wave, but the treatment for hypovolemia can dramatically worsen the congestive heart failure secondary to dilatative cardiomyopathy.

When a patient presents with some form of shock, immediate vascular access is of paramount importance. Place a large-bore peripheral or central venous catheter for the infusion of crystalloid or colloid fluids, blood component therapy, and drugs. Monitor the patient's cardiopulmonary status (by ECG), blood pressure, oxygen saturation (as determined by pulse oximetry or arterial blood gas analyses), hematocrit, BUN, and glucose. Ancillary diagnostics, including thoracic and abdominal radiography, urinalysis, serum biochemistry profile, coagulation tests, complete blood count, abdominal ultrasound, and echocardiography, should be performed as determined by the individual patient's needs and the type of shock.

MANAGEMENT OF THE SHOCK PATIENT

THE RULE OF TWENTY

The following list, called the "Rule of Twenty," is a guideline for case management of the shock patient. Consideration of each aspect of the Rule of Twenty on a daily basis ensures

TABLE 1 - 57 Clinical Signs of Shock Syndrome

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Please refer to the printed publication.

that major organ systems are not overlooked. The list also provides a means to integrate and relate changes in different organ systems functions with one another.*

1. Fluid balance

The treatment of hypovolemic and septic shock requires the placement of large-bore intravenous catheters in peripheral and central veins. If vascular access cannot be obtained percutaneously or by cutdown methods, intraosseous catheterization should be considered. Once vascular access is achieved, rapidly administer large volumes of crystalloid or colloid fluids. As a rule of thumb, administer $\frac{1}{4}$ of a calculated shock dose of fluids—that is, $\frac{1}{4} \times (90 \text{ mL/kg/hour})$ in dogs and $\frac{1}{4} \times (44 \text{ mL/kg/hour})$ in cats) of a balanced crystalloid fluid (Normosol-R, Plasmalyte-M, lactated Ringer's solution, or 0.9% sterile saline). Reassess the patient's perfusion parameters (heart rate, capillary refill time, blood pressure, urine output) on a continual basis to direct further fluid therapy. Synthetic colloid fluids (hetastarch, dextran 70, or Oxyglobin) can also be administered in the initial resuscitation from shock. A guideline is to administer 5 to 10 mL/kg of hetastarch or dextran as a bolus over 10 to 15 minutes and then reassess perfusion parameters. Hypertonic saline (0.7% NaCl, 4 mL/kg) can be used in cases of hemorrhagic shock to temporarily restore intravascular fluid volume by drawing fluid from the interstitial space. because this type of fluid resuscitation is short-lived, hypertonic saline should always be used with another crystalloid or colloid fluid, and it should not be used in patients with interstitial dehydration. If hemorrhagic shock is present, the goal should be to return a patient's blood pressure to normal (not supraphysiologic) levels (i.e., systolic pressure 90-100 mm Hg, diastolic pressure >40 mm Hg, and mean arterial pressure ≥ 60 mm Hg) to avoid iatrogenically causing clots to fall off and hemorrhage to re-start.

In critically ill patients, fluid loss can be measured in the form of urine, vomit, diarrhea, body cavity effusions, and wound exudates. Additionally, insensible losses (those that cannot be readily measured from sweat, panting, and cellular metabolism) constitute 20 mL/kg/day. Measurement of fluid "ins and outs" in conjunction with the patient's central venous pressure, hematocrit, albumin, and colloid oncotic pressure can help guide fluid therapy (see also section on Fluid Therapy).

*From Purvis D, Kirby R. Systemic inflammatory response syndrome: septic shock. *Vet Clin North Amer Small Anim Pract* 24(6):1225-1247, 1994.

Kirby R: Septic shock. In Bonagura JD, editor: *Kirk's current veterinary therapy XII*. Philadelphia, 1995, WB Saunders.

TABLE 1-58 Sympathomimetic Drugs Used to Treat Cardiogenic Shock

Drug	Receptor activity	Dosage (IV)
Dopamine	DA ₁ , DA ₂ , α^{+++} , β^{+++}	5-25 $\mu\text{g/kg/minute}$ (blood pressure support)* 1-5 $\mu\text{g/kg/minute}$ (renal afferent diuresis)
Dobutamine	α^+ , β^{+++}	3-20 $\mu\text{g/kg/minute}^*$ (blood pressure support, positive inotrope)
Norepinephrine	α^{+++} , β^+	0.05-0.3 mg/kg/minute; 0.01-0.02 mg/kg
Phenylephrine	α^{+++} , β^0	0.05-0.2 mg/kg
Epinephrine	α^{+++} , β^{+++}	0.02-0.5 mg/kg, 0.05-0.2 mg/kg/minute

+++ , Strong receptor activity; 0, no receptor activity; +, weak receptor activity.

*Monitor for tachyarrhythmias at higher doses.

2. Blood pressure

Maintenance of normotension is necessary for adequate oxygen delivery to meet cellular energy demands. Blood pressure can be measured using direct arterial catheterization, or through indirect means such as Doppler plethymography or oscillometric methods. The systolic pressure should remain at or greater than 90-100 mm Hg at all times. The diastolic pressure is very important, too, as it constitutes two thirds of the mean arterial pressure; it must be greater than 40 mm Hg for coronary artery perfusion. The mean arterial pressure should be greater than 60 mm Hg for adequate tissue perfusion.

If fluid resuscitation and pain management are not adequate in restoring blood pressure to normal, vasoactive drugs including positive inotropes and pressors should be considered (Table 1-58).

In cases of cardiogenic shock, vasodilator drugs (Table 1-59) can be used to decrease vascular resistance and afterload. Low-dose morphine (0.05 mg/kg, IV, IM) dilates splanchnic vessels and helps reduce pulmonary edema. Furosemide (1 mg/kg/hour) also can dilate pulmonary vasculature and potentially reduce edema fluid formation in cases of ARDS.

3. Heart rate, rhythm, contractility, and pulse quality

Cardiac output is a function of both heart rate and stroke volume. Stroke volume or (the amount of blood that the ventricle pumps in 1 minute) is affected by preload, afterload, and contractility. During hypovolemic shock, there is a fall in cardiac preload due to a decrease in circulating blood volume. During septic and cardiogenic shock, there is a decrease in contractility secondary to inherent defects of the myocardium or due to the negative inotropic effects of inflammatory cytokines such as TNF-alpha, myocardial depressant factor, IL-1, and IL-10 released during sepsis and systemic inflammation. Afterload also may be increased because of the compensatory mechanisms and neuro-humoral activation of the renin-angiotensin-aldosterone axis in hypovolemic or cardiogenic shock. As heart rate increases to compensate for a decline in cardiac output, myocardial oxygen demand increases and diastolic filling time becomes shorter. Because the coronary arteries are perfused during diastole, coronary perfusion can be impaired, and myocardial lactic acidosis can develop, causing a further decline in contractility. In addition to lactic acidosis, acid-base and electrolyte abnormalities, inflammatory cytokines, direct bruising of the myocardium from trauma, and areas of ischemia can further predispose the patient to ventricular or atrial dysrhythmias.

Cardiac dysrhythmias should be controlled whenever possible. Treatment of bradycardia should be directed at treating the underlying cause. Administer anticholinergic drugs such as atropine (0.04 mg/kg IM) or glycopyrrolate (0.02 mg/kg IM) as necessary. In cases of third-degree or complete atrioventricular (AV) block, administer a pure beta-agonist such as isoproterenol (0.04-0.08 $\mu\text{g/kg/minute}$ IV CRI, or 0.4 mg in 250 mL of 5% dextrose in water IV slowly). Perform passive rewarming if the patient is hypothermic.

TABLE 1-59 Drugs and Doses Used to Induce Vasodilatation

Drug	Mechanism of action	Dose and method of administration (onset, peak, duration)	Potential adverse effects
Captopril	Angiotensin-converting enzyme inhibitor	0.5-2 mg/kg PO tid	Azotemia
Enalapril	Angiotensin-II-converting enzyme inhibitor	0.25-0.5 mg/kg/PO q12-24h	Azotemia
Hydralazine	Direct arteriolar smooth muscle relaxant; little effect on venous capacitance vessels	0.2-0.5 mg/kg (10-20 minutes; 10-80 minutes, 2-8 hour)	Blood dyscrasias, neuritis with prolonged use
Lisinopril	Angiotensin-II-converting enzyme inhibitor		
Morphine	Splanchnic capacitance vessel dilatation	0.025-0.05 mg/kg IV, IM, SQ	Vomiting
Nitroglycerine paste	Venous and some arteriolar dilatation	1/4 to 3/4 inch topically on skin every 8 hours	Tolerance after 48 hours
Prazosin	α -Receptor blockade; arteriolar and venous dilatation	1-3 mg/kg PO bid	Anorexia, vomiting, diarrhea
Sodium nitroprusside	Direct arteriolar and venular dilatation	0.5-10 μ g/kg/minute CRI; dilute in 5% dextrose in water Intravenous line with continuous blood pressure monitoring (immediately; 1 minute; 2 minutes)	Hypotension, tolerance, cyanide toxicity at higher doses, avoid in hepatic or renal failure; thiocyanate accumulation (disorientation); is light sensitive and must be covered in foil and not kept for longer than 4 hours

Correct any underlying electrolyte abnormalities such as hyperkalemia and hypo- and hypermagnesemia.

Treat ventricular dysrhythmias such as multifocal premature ventricular contractions (PVCs), sustained ventricular tachycardia >160 beats per minute, and R on T phenomenon (the T wave of the preceding beat occurs superimposed on the QRS complex of the next beat, and there is no return to isoelectric shelf), or if runs of ventricular tachycardia cause a drop in blood pressure. Intravenous lidocaine and procainamide are the first drugs of choice for ventricular dysrhythmias. Supraventricular tachycardia can impair cardiac output by impairing diastolic filling time. Control supraventricular dysrhythmias with calcium channel blockers, beta-adrenergic blockers, or quinidine (Table 1-60).

TABLE 1 - 60 Antiarrhythmic Drugs of Choice Used for the Treatment of Ventricular and Supraventricular Tachycardias

Drug	Mechanism of action	Dose
Lidocaine	Fast sodium channel inhibition	1-4 mg/kg IV slowly, then 50-100 µg/kg/minute (dog); 0.25-1.0 mg/kg IV (cat)*
Procainamide	Fast sodium channel inhibition	1-8 mg/kg IV, † then 20-40 µg/kg/minute; 6-20 mg/kg PO tid
Tocainide	Fast sodium channel inhibition	5-20 mg/kg PO Q8h‡ (dog)
Quinidine	Fast sodium channel inhibition	6-10 mg/kg PO qid
Propranolol	β-Adrenergic blocker	0.02-0.06 mg/kg IV; 0.02-1.0 mg/kg PO
Esmolol	β-Adrenergic blocker	0.5 mg/kg IV, then 50-200 µg/kg/minute IV CRI
Verapamil	Slow calcium channel blocker	0.01-1 mg/kg IV; 5-10 mg/kg PO q8h
Diltiazem	Calcium channel blocker	0.25 mg/kg IV, 0.5-1.5 mg/kg PO q8h (dogs) 1.75-2.5 mg/kg PO q8h
Pimobendan	Phosphodiesterase inhibition Positive inotrope	0.1-0.3 mg/kg PO q12h

*Use caution with lidocaine in cats because of neurotoxicity and seizures.

† Monitor for hypotension.

‡Is not to be used for more than 2 weeks due to idiosyncratic blindness.

4. Albumin

Albumin can decrease as a result of loss from the gastrointestinal tract, urinary system, and wound exudates, or into body cavity effusions. Albumin synthesis can decrease during various forms of shock due to a preferential increase in hepatic acute phase protein synthesis. Serum albumin contributes 80% of the colloid oncotic pressure of blood, in addition to its important roles as a free radical scavenger at sites of inflammation and as a drug and hormone carrier. Albumin levels <2.0 g/dL have been associated with an increase in morbidity and mortality in human and veterinary patients. Administer fresh frozen plasma (20 mL/kg) or concentrated human albumin (2 mL/kg of 25% solution) to maintain serum albumin ≥2.0 g/dL. Additional oncotic support can be in the form of synthetic colloids, as indicated.

5. Oncotic pressure

Colloid oncotic pressure within the intravascular and interstitial spaces contributes to fluid flux. Oncotic pressure can be measured with a colloid osmometer. Normal oncotic pressure is 15 mm Hg. In cases of sepsis and SIRS, increased vascular permeability increases the tendency for leakage of fluids into the interstitial spaces. Colloids that can be administered until the source of albumin loss resolves include the synthetic colloids hetastarch and dextran 70 (20-30 mL/kg/day), synthetic hemoglobin-based oxygen carriers (oxyglobin, 3-7 mL/kg/day), concentrated human albumin (25% albumin, 2 mL/kg), and plasma (20 mL/kg).

6. Oxygenation and ventilation

Oxygenation and ventilation can be evaluated by arterial blood gas analysis or by the noninvasive means of pulse oximetry and capnometry (see sections on Pulse Oximetry and Capnometry). Oxygen delivery can be impaired in cases of hypovolemic shock because of hemorrhage and anemia, and thus a decrease in functional capacity to carry oxygen, and

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in cases of cardiogenic shock as a result of impaired ability to saturate hemoglobin due to pulmonary edema in the lungs, or decrease in cardiac output. In septic shock, decreases in cardiac output due to inflammatory cytokines and a decrease in cellular oxygen extraction can lead to lactic acidosis. Increased cellular metabolism and decreases in respiratory function can lead to respiratory acidosis as CO_2 increases.

Administer supplemental oxygen as flow-by, nasal or nasopharyngeal catheter, oxygen hood, or oxygen cage. Supplemental oxygen should be humidified, and delivered at 50-100 mL/kg/minute. If oxygenation and ventilation are so impaired that the PaO_2 remains <60 mm Hg with the patient on supplemental oxygen, a $\text{PaCO}_2 >60$ mm Hg, or severe respiratory fatigue, develops, and mechanical ventilation should be considered.

7. Glucose

Glucose is a necessary fuel source for red blood cells and neuronal tissues, and serum glucose should be maintained within normal reference ranges. Glucose supplementation can be administered as 2.5-5% solutions in crystalloid fluids, or in parenteral and enteral nutrition products.

8. Acid-base and electrolyte and lactate status

Arterial and venous pH can be measured by performing blood gas analyses. Decrease in tissue perfusion, impaired oxygen delivery, and decreased oxygen extraction in the various forms of shock can lead to anaerobic metabolism and metabolic acidosis. In most cases, improving tissue perfusion and oxygen delivery with crystalloid and colloid fluids, supplemental oxygen, and inotropic drugs will help normalize metabolic acidosis. Serial measurements of serum lactate (normal, <2.5 mmol/L) can be used as a guide to evaluate the tissue response to fluid resuscitative efforts.

Serum electrolytes often become severely deranged in shock states. Serum potassium, magnesium, sodium, chloride, and total and ionized calcium should be maintained within normal reference ranges.

If metabolic acidosis is severe, sodium bicarbonate can be administered by calculating the formula

$$\text{Base deficit} \times 0.3 \times \text{body weight in kg} = \text{mEq bicarbonate to administer}$$

Because iatrogenic metabolic alkalosis can occur, a conservative approach is to administer $\frac{1}{4}$ of the calculated dose and then recheck the patient's pH and bicarbonate levels. If the base excess is unknown, sodium bicarbonate can be administered in incremental doses of 1 mEq/kg until the pH is above 7.2. Complications associated with bicarbonate therapy include iatrogenic hypocalcemia, metabolic alkalosis, paradoxical cerebrospinal fluid acidosis, hypotension, restlessness, and death.

9. Coagulation

Massive trauma, neoplasia, sepsis, and systemic inflammation can all lead to coagulation abnormalities, including disseminated intravascular coagulation (DIC). Cage-side coagulation monitors are available for daily measurement of prothrombin time (PT), activated partial thromboplastin time (APTT), and platelet counts. Fibrin degradation products (fibrin split products) become elevated in DIC, trauma, hepatic disease, and surgery. Coagulation proteins (clotting factors) and antithrombin often are lost with other proteins in hypoproteinemia or are consumed when microclots are formed and then dissolved. Antithrombin levels can be measured by commercial laboratories. Antithrombin and clotting factors can be replenished in the form of fresh frozen plasma transfusions. A more sensitive and specific test for DIC is the detection of D-dimers, which can be measured by commercial laboratories.

Treatment for DIC involves treatment and resolution of the underlying disease and administration of antithrombin and clotting factors in the form of fresh frozen plasma (20 mL/kg) and heparin (unfractionated, 50-100 units/kg SQ tid; fractionated [Lovenox], 1 mg/kg SQ bid).

10. Mentation

Monitor the patient for changes in mental status, including stupor, coma, decreased ability to swallow and protect the airway, and seizures. Elevation of the patient's head can help to protect the airway and decrease the risk of increased intracranial pressure. Serum glucose should be maintained within normal levels to prevent hypoglycemia-induced seizures.

11. Red blood cell and hemoglobin concentration

One of the major components of oxygen delivery is the binding to hemoglobin. Packed cell volume must be kept above 20-30% for adequate cellular oxygen delivery. Acid-base status can adversely affect oxygen offloading at the tissue level if metabolic or respiratory alkalosis is present. Oxygen-carrying capacity and hemoglobin levels can be increased with administration of RBC component therapy or with hemoglobin-based oxygen carriers.

12. Renal function

Monitoring of renal function includes daily measurement of BUN, creatinine, and urine output. Normal urine output in a hydrated euvoletic patient is 1-2 mL/kg/hour. Fluid ins and outs should be measured in cases of suspected oliguria or anuria. In patients with oliguria or anuria, furosemide can be administered as a bolus (4-8 mg/kg) or by constant rate infusion (CRI)(0.66-1 mg/kg/hour). Mannitol should also be administered (0.5-1 g/kg over 10 to 15 minutes). Dopamine (1-5 µg/kg/minute CRI) can be administered to dilate renal afferent vessels and improve urine output.

13. White blood cell count, immune function, antibiotic dose and selection

The patient's white blood cell count may be elevated, normal, or decreased, depending on the type of shock. The decision to administer antibiotics should be made on a daily basis. Superficial or deep *Staphylococcus* or *Streptococcus* infection usually can be treated with a first-generation cephalosporin (cefazolin, 22 mg/kg IV tid). If a known source of infection is present, administer a broad-spectrum antibiotic (cefoxitin, 22 mg/kg IV tid; ampicillin, 22 mg/kg qid, or enrofloxacin, 5-10 mg/kg once daily) pending results of culture and susceptibility testing. If broader anaerobic coverage is required, metronidazole (10 mg/kg IV tid) should be considered. Gentamicin (3-5 mg/kg IV once daily) is a good choice for gram-negative sepsis, provided that the patient is well hydrated and has normal renal function. Ideally, patients receiving any aminoglycoside antibiotic should have a daily urinalysis to check for renal tubular casts that signify renal damage.

14. Gastrointestinal motility and integrity

In dogs, the gut is the shock organ. Impaired gastrointestinal motility and vomiting should aggressively be treated with antiemetics and promotility drugs (dolasetron, 0.6 mg/kg IV once daily, and metoclopramide, 1-2 mg/kg/day IV CRI). Metoclopramide is contraindicated in cases of suspected gastrointestinal obstruction. Histamine-receptor blockers such as famotidine (0.5 mg/kg bid IV) and ranitidine (0.5 to 2 mg/kg IV bid, tid) or proton-pump inhibitors (omeprazole, 0.5-1 mg/kg PO once daily) can be administered for esophagitis. Administer sucralfate (0.25-1 g PO tid) to treat gastric ulceration. If the gastrointestinal barrier function is diminished due to poor perfusion, infection, or inflammation, administer broad-spectrum antibiotics such as ampicillin (22 mg/kg IV qid) to prevent gastrointestinal bacterial translocation.

15. Drug doses and metabolism

The course of drug therapy should be reviewed daily and the patient should be monitored for potential drug interactions. For example, metoclopramide and dopamine, working at the same receptor, can effectively negate the effects of each other. Cimetidine, a cytochrome P450 enzyme inhibitor, can decrease the metabolism of some drugs. Drugs that are avidly protein-bound may have an increase in unbound fraction with concurrent hypoalbuminemia or when hypoalbuminemia is present. Decreased renal function may impair the renal clearance of some drugs, requiring increased dosing interval or decreased dose.

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16. Nutrition

Nutrition is of utmost importance in any critically ill patient. Patients with septic shock may become hypermetabolic and require supraphysiologic nutrient caloric requirements, while others may actually become hypometabolic. Enteral nutrition is preferred, whenever possible, because enterocytes undergo atrophy without luminal nutrient stimulation. A variety of enteral feeding tubes can be placed, depending on what portion of the gut is functional, to provide enteral nutrition in an inappetent patient. Loss of gastrointestinal mucosal barrier function may predispose the patients to the development of bacterial translocation and may contribute to sepsis. If enteral nutrition is impossible because of protracted vomiting or gastrointestinal resection, glucose, lipid, and amino acid products are available that can be administered parenterally to meet nutrient needs until the gastrointestinal tract is functioning and the patient can be transitioned to enteral nutrition.

17. Analgesia and pain control

Assessment of pain in animals in shock can be challenging. Pain can result in the release of catecholamines and glucocounterregulatory hormones that can impair nutrient assimilation and lead to negative nitrogen balance, impaired wound healing, and immunocompromise. In any animal determined to be in pain, analgesic drugs should be administered to control pain and discomfort at all times. Opioids are cardiovascularly friendly, and their effects can easily be reversed with naloxone if adverse effects such as hypotension and hypoventilation occur.

18. Nursing care and patient mobilization

If the patient is nonambulatory, rotate the animal from side to side every 4 to 6 hours to prevent lung atelectasis. Passive range-of-motion exercises and deep muscle massage should be performed to increase tissue perfusion, decrease dependent edema, and prevent disuse atrophy. Animals should be kept completely dry on soft, padded bedding to prevent the development of decubital ulcers.

19. Wound care/bandage care

All bandages, wound sites, and catheter sites should be checked daily for the presence of swelling, erythema, and pain. Soiled bandages should be changed to prevent strike-through and contamination of the underlying catheter or wound.

20. TLC (tender loving care)

Hospitalization can be a stressful experience for patient and client alike. Allowing brief visits and walks outside in the fresh air can improve a patient's temperament and decrease stress. The preemptive use of analgesic drugs on a regular schedule (not PRN) should be used to prevent pain *before* it occurs. Pain decreases the patient's ability to sleep. Lack of sleep can promote further stress and impaired wound healing.

OTHER CONSIDERATIONS AND CONTROVERSIES IN SHOCK THERAPY**Glucocorticosteroids and antiprostaglandins**

The use of glucocorticosteroids and antiprostaglandins in shock therapy remains a topic of wide controversy. Although the use of these agents potentially may stabilize membranes, decrease the absorption of endotoxin, and decrease prostaglandin release, the routine use of glucocorticosteroids and antiprostaglandins can decrease renal perfusion and gastrointestinal blood flow, promoting gastrointestinal ulceration and impaired renal function. The administration of supraphysiologic levels of glucocorticosteroids in patients in any type of shock can increase sodium and water retention, depress cellular immune function, and impair wound healing. In clinical studies of small animal patients, the routine use of glucocorticosteroids and antiprostaglandins has not demonstrated definite improved survival. The risks of therapy do outweigh the anecdotal reported benefits, and therefore the empiric use of glucocorticosteroids and antiprostaglandins in any shock patient is

absolutely contraindicated. The administration of glucocorticosteroids to patients with cardiac disease has been shown to promote sodium and water retention and can actually predispose to the development of congestive heart failure.

Additional Reading

- Brady CA, Otto CM: Systemic inflammatory response syndrome, sepsis, and multiple organ dysfunction. *Vet Clin North Am Small Anim Pract* 31(6):1147-1162, 2000.
- Buston R: Treatment of congestive heart failure. *J Small Anim Pract* 44(11):516, 2003.
- Chan DL, Rozanski EA, Freeman LM, Rush JE: Colloid osmotic pressure in health and disease. *Compend Contin Educ Pract Vet* 23(10):896-904, 2001.
- Cote E: Cardiogenic shock and cardiac arrest. *Vet Clin North Am Small Anim Pract* 31(6):1129-1145, 2001.
- DeLaforcade AM, Freeman LM, Shaw SP, Brooks MB, et al: Hemostatic changes in dogs with naturally occurring sepsis. *J Vet Intern Med* 17(5):674-679, 2003.
- Johnson V, Gaynor A, Chan DL, et al: Multiple organ dysfunction syndrome in humans and dogs. *J Vet Emerg Crit Care* 14(3):158-166, 2004.
- Lagutchik MS, Ogilvie GK, Hackett TB, et al: Increased lactate concentrations in ill and injured dogs. *J Vet Emerg Crit Care* 8(2):117-127, 1998.
- Mazzaferro EM, Rudloff E, Kirby R: The role of albumin in health and disease. *J Vet Emerg Crit Care* 12(2):113-124, 2002.
- Moore KE, Murtaugh RJ: Pathophysiologic characteristics of hypovolemic shock. *Vet Clin North Am Small Anim Pract* 31(6):1115-1128, 2001.
- Okano S, Yoshida M, Fukuahima U, et al: Usefulness of systemic inflammatory response syndrome criteria as an index for prognosis judgement. *Vet Rec* 150(8):245-246, 2002.
- Otto CM: Sepsis. In Wingfield WE, Raffe M (eds): *The Veterinary ICU Book*. Teton New Media, Jackson, Wyo, 2001.
- Rossmesl JH: Current principles and application of D-dimer analysis in small animal practice. *Vet Med* 98(3):224-234, 2003.
- Rozanski E, Rondeau M: Choosing fluids in traumatic hypovolemic shock: the role of crystalloids, colloids and hypertonic saline. *J Am Anim Hosp Assoc* 38(6):499-501, 2002.
- Rudloff E, Kirby R: Colloid and crystalloid resuscitation. *Vet Clin North Am Small Anim Pract* 31(6):1207-1229, 2001.

THROMBOEMBOLISM: SYSTEMIC

Systemic thromboembolism is most commonly recognized in cats with cardiomyopathies (hypertrophic, restrictive, unclassified, and dilatative) but can also occur in dogs with hyperadrenocorticism, disseminated intravascular coagulation (DIC), systemic inflammatory response syndrome (SIRS), protein-losing enteropathy and nephropathy, and tumors affecting the aorta and vena cava. Thrombosis occurs through a complex series of mechanisms when the components of Virchow's triad (hypercoagulable state, sluggish blood flow, and vascular endothelial injury or damage) are present. In cats, blood flow through a severely stretched left atrium is a predisposing factor to the development of clots and thromboembolism.

The most common site of embolism is the aortic bifurcation, or "saddle thrombus." Other, less common locations of thromboembolism include the forelimbs, kidneys, gastrointestinal tract, and cerebrum. Diagnosis usually is made based on clinical signs of cool extremities, the presence of a cardiac murmur or gallop rhythm, auscultation of pulmonary crackles resulting from pulmonary edema, acute pain or paralysis of one or more peripheral extremities, respiratory distress, and pain and lack of a palpable pulse in affected limbs. The affected nailbeds and paw pads are cyanotic, and nails do not bleed when cut with a nail clipper.

Client education is one of the most important aspects of emergency management of the patient with thromboembolic disease. Concurrent congestive heart failure (CHF) occurs in 40% to 60% of cats with arterial thromboembolism. More than 70% of cats are euthanized during the initial thromboembolic event because of the poor long-term prognosis and the high risk of recurrence within days to months after the initial event, even with aggressive therapy. Although the long-term prognosis varies from 2 months to 2 years after initial

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diagnosis and treatment, in the majority of cats thromboembolic disease recurs within 9 months. Rectal temperature hypothermia and bradycardia on presentation are negative prognostic indicators.

Immediate treatment of a patient with CHF and thromboembolic disease involves management of the CHF with furosemide, oxygen, and vasodilators (nitroglycerine paste, morphine, nitroprusside). Additional management includes analgesia (butorphanol, 0.1-0.4 mg/kg IV, IM) and prevention of further clot formation. Aspirin (10 mg/kg PO q48h) is beneficial because of its antiplatelet effects. Heparin works in conjunction with antithrombin to prevent further clot formation (100-200 units/kg IV, followed by 250-300 units/kg SQ q8h in cats, and 100-200 units/kg SQ q8h in dogs). Acepromazine can cause peripheral vasodilation and decreased afterload but also can promote hypotension in a patient with concurrent CHF. Acepromazine (0.01-0.02 mg/kg SQ) should be used with extreme caution, if at all.

Thrombolytic therapy can also be attempted, but in most cases is not without risk, and may be cost-prohibitive for many clients. Streptokinase (90,000 units IV over 30 minutes and then 45,000 units/hour IV CRI for 3 hours) was administered with some success in cats; however, many died of hyperkalemia or other complications during the infusion. Tissue plasminogen activator (0.25-1 mg/kg/hour IV CRI, up to 10 mg/kg total dose, to effect) has been used with some success but is cost-prohibitive for most clients. Side effects of thrombolytic therapy include hyperkalemia with reperfusion and hemorrhage.

In cats, the primary cause of arterial thromboembolism is cardiomyopathy. Once an animal is determined to be stable enough for diagnostic procedures, lateral and DV thoracic radiographs and an echocardiogram should be performed. Ultrasound of the distal aorta and renal arteries should also be performed to determine the location of the clot and help establish the prognosis.

Other diagnostic procedures to evaluate the presence and cause of thromboembolism include a complete blood count, serum biochemistry profile, urinalysis (to rule out protein-losing nephropathy), urine protein:creatinine ratio, antithrombin levels, ACTH stimulation test (to rule out hyperadrenocorticism), heartworm antigen test (in dogs), thyroid profile (to rule out hyperthyroidism in cats, and hypothyroidism in dogs), thoracic radiographs, arterial blood gas analyses, coagulation tests, and Coombs' test. Selective and nonselective angiography can also be performed to determine the exact location of the thrombus.

Long-term management of thromboembolism involves management of the underlying disease process and preventing further clot formation. Begin therapy with heparin until the APTT becomes prolonged 1.5 times; then administer warfarin (0.06-0.09 mg/kg/day). Monitoring therapy based on prothrombin time and the international normalized ratio (INR, 2.0-4.0) is recommended. Low-dose aspirin (5-10 mg/kg q48h) also has been recommended. Physical therapy with warm water bathing, deep muscle massage, and passive range-of-motion exercises should be performed until the patient regains motor function. Future therapy may involve the use of platelet receptor antagonists to prevent platelet activation and adhesion.

Additional Reading

- Good LI, Manning AM: Thromboembolic disease: predispositions and management. *Comp Contin Educ Pract Vet* 25(9):660-674, 2003.
- Marks SL: Systemic arterial thromboembolism. In Wingfield WE (ed): *Veterinary Emergency Medicine Secrets*. Hanley and Belfus, Philadelphia, 2001.
- Moore KE, Morris N, Dhupa N, et al: Retrospective study of streptokinase administration in 46 cats with arterial thromboembolism. *J Vet Emerg Crit Care* 10(4):245-257, 2000.
- Schoeman JP: Feline distal aortic thromboembolism: a review of 44 cases (1990-1998). *J Feline Med Surg* 1:221-231, 1999.
- Smith SA, Tobias AH: Feline arterial thromboembolism: an update. *Vet Clin North Am Small Anim Pract* 34(5):1245-1271, 2004.
- Smith SA, Tobias AH, Jacob KA, et al: Arterial thromboembolism in cats: acute crises in 127 cases (1992-2001) and long-term management with low-dose aspirin in 24 cases. *J Vet Intern Med* 17(1):73-83, 2003.

URINARY TRACT EMERGENCIES

AZOTEMIA

Azotemia occurs when 75% or more of the nephrons are nonfunctional. The magnitude of the azotemia alone cannot be used to determine whether the azotemia is prerenal, renal, or postrenal in origin, or whether the disease process is acute or chronic, reversible or irreversible, progressive or nonprogressive. Before beginning treatment for azotemia, the location or cause of the azotemia must be identified. Take a thorough history and then perform a physical examination. Obtain blood and urine samples before initiating fluid therapy, for accurate assessment of the location of the azotemia.

For example, an azotemic animal with a history of vomiting and diarrhea that appears clinically dehydrated on physical examination, normally should have a concentrated urine specific gravity (>1.045) reflecting the attempt to conserve fluid. If this level is found, the azotemia is much less likely to be renal in origin, and the azotemia will likely resolve after rehydration.

If, however, the urine specific gravity is isosthenuric or hyposthenuric (1.007-1.015) in the presence of azotemia and dehydration, primary intrinsic renal insufficiency is likely present. If the azotemia resolves with fluid therapy, the patient has prerenal and primary renal disease. If the azotemia does not resolve after rehydration, the patient has prerenal and primary renal failure. Dogs with hypoadrenocorticism can have both prerenal and primary renal disease secondary to the lack of mineralocorticoid (aldosterone) influence on the renal collecting duct and renal interstitial medullary gradient. Medullary washout can occur, causing isosthenuric urine in the presence of dehydration from vomiting and diarrhea. The patient often has azotemia due to fluid loss (dehydration and urinary loss) and gastric or intestinal hemorrhage (elevated BUN). The prerenal component will resolve with treatment with glucocorticoids and crystalloid fluids, but the renal component may take several weeks to resolve, until the medullary concentration gradient is reestablished with the treatment and influence of mineralocorticoids. Drugs such as corticosteroids and diuretics can influence renal tubular uptake and excretion of fluid, and cause a prerenal azotemia and isosthenuric urine in the absence of primary renal disease.

Treatment of azotemia includes calculation of the patient's dehydration estimate and maintenance fluid volumes, and administering that volume over the course of 24 hours. Identify and treat underlying causes of prerenal azotemia (shock, vomiting, diarrhea). Monitor urine output closely. Once a patient is euvolemic, oliguria is defined as urine output <1 - 2 mL/kg/hour. Urine output should return to normal in patients with prerenal azotemia as rehydration occurs. If a patient remains oliguric after rehydration, consider the possibility of oliguric acute intrinsic renal failure, and administer additional fluid therapy based on the patient's urine output, body weight, central venous pressure, and response to other medical therapies.

PRERENAL AZOTEMIA

Prerenal azotemia is caused by conditions that decrease renal perfusion, including hypovolemic shock, severe dehydration, hypoadrenocorticism, congestive heart failure, cardiac tamponade, cardiac dysrhythmias, and hypotension. Once renal perfusion is restored, the kidneys can resume normal function. Glomerular filtration rate decreases when the mean arterial blood pressure falls to less than 80 mm Hg in a patient with normal renal autoregulation. Renal autoregulation can be impaired in some diseases. Passive reabsorption of urea from the renal tubules can occur during states of low tubular flow (dehydration, hypotension) even if glomerular filtration is not decreased. If renal hypoperfusion is not quickly restored, the condition can progress from prerenal disease to acute intrinsic renal failure. Prerenal and renal azotemia can coexist in animals with primary renal disease, as a result of vomiting and ongoing polyuria in the absence of any oral fluid intake. The treatment of prerenal azotemia consists of rehydration, antiemetic therapy, and treatment of the underlying cause of vomiting, diarrhea, or third spacing of fluids.

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ACUTE INTRINSIC RENAL FAILURE

Acute intrinsic renal failure is characterized by an abrupt decline in renal function to the extent that azotemia and an inability to regulate solute and fluid balance. Patients with acute intrinsic renal failure may be oliguric or polyuric, depending on the cause and state of renal failure. In small animals, the most common causes of acute intrinsic renal failure are renal ischemia and toxins.

There are three phases of acute intrinsic renal failure: induction, maintenance, and recovery. During the induction phase, some insult (ischemia or toxin) to the kidneys occurs, leading to a defective concentrating mechanism, decreased renal clearance of nitrogenous waste (azotemia), and polyuria or oliguria. If treatment is initiated during the induction phase, progression to the maintenance phase potentially can be stopped. As the induction phase progresses, there is worsening of the urine-concentrating ability and azotemia. Renal tubular epithelial cells and renal tubular casts can be seen on examination of the urine sediment. Glucosuria may be present.

The maintenance phase of acute intrinsic renal failure occurs after a critical amount of irreversible nephron injury. Correction of the azotemia and removal of the cause of the problem do not result in return to normal function. In patients with oliguria, the extent of nephron damage is greater than that observed in patients with polyuria. The maintenance phase may last for several weeks to months. Recovery of renal function may or may not occur, depending on the extent of injury. The most serious complications (overhydration and hyperkalemia) are observed in patients with oliguria.

The recovery phase occurs with sufficient healing of damaged nephrons. Azotemia may resolve, but concentrating defects may remain. If the patient was oliguric in the maintenance phase, a marked diuresis develops during the recovery phase that may be accompanied by fluid and electrolyte losses. This phase may last for weeks to months.

Treatment of acute intrinsic renal failure consists of determining the cause and ruling out obstruction or uroabdomen whenever possible. A careful history can sometimes determine whether there has been exposure to nephrotoxic drugs, chemicals, or food items. If ingestion or exposure to a toxic drug, chemical, or food occurred recently (within 2 to 4 hours), induce emesis with apomorphine (0.04 mg/kg IV). Next, administer activated charcoal either orally or via stomach tube, to prevent further absorption of the toxin. Obtain blood and urine samples for toxicologic analysis (e.g., ethylene glycol) and to determine whether azotemia or abnormalities in the urine sediment exist. (See section on Ethylene Glycol, Grapes and Raisins, and Nonsteroidal Antiinflammatory Drugs). Obtain a complete blood count, biochemical profile, and urinalysis to determine the presence of signs of chronic renal failure, including polyuria, polydipsia, and nonregenerative anemia. Radiographs and abdominal ultrasound can help in determining the chronicity of renal failure. Normal renal size is 2.5-3.5 times the length of L2 in dogs and 2.4-3.0 times the length of L2 in cats. Monitor the patient's body weight at least twice a day to avoid overhydration.

Also monitor urine output; normal output is 1-2 mL/kg/hour. In cases of polyuric renal failure, massive fluid and electrolyte losses can occur. Place a urinary catheter for patient cleanliness and to facilitate urine quantitation. Measure fluid ins and outs (see section on Fluid Therapy). After the patient has been rehydrated, the amount of fluids administered should equal maintenance and insensible needs plus the volume of urine produced each day. If a urinary catheter cannot be placed or maintained, serial body weight measurements and central venous pressure should be used to monitor the patient's fluid balance and prevent overhydration.

If the patient is oliguric (urine output <1-2 mL/kg/hour), pharmacologic intervention is necessary to increase urine output. First, administer furosemide (2-4 mg/kg or 0.66 mg/kg/hour IV CRI). Repeat bolus doses of furosemide if there is no response to initial treatment. If necessary, administer low-dose dopamine (3-5 µg/kg/minute IV CRI) to increase renal afferent dilatation and renal perfusion. Dopamine and furosemide may be synergistic if administered together. If dopamine and furosemide therapy is ineffective, administer mannitol (0.25-0.5 g/kg IV) *once only*. If polyuria is present, management is

simplified because of the decreased risk of overhydration. If oliguria cannot be reversed, monitor the central venous pressure, body weight, and respiratory rate and effort, auscultate for crackles, and examine the patient carefully for signs of chemosis and the presence of serous nasal discharge.

Correct hyperkalemia with sodium bicarbonate (0.25-1.0 mEq/kg IV) or with insulin (0.25 units/kg) plus dextrose (1 g/unit of insulin IV, followed by 2.5% dextrose IV CRI). Treat severe metabolic acidosis ($\text{pH} < 7.2$ or $\text{HCO}_3^- < 12$ mEq/L) with sodium bicarbonate. If anuria develops or oliguria is irreversible despite this therapy, begin peritoneal dialysis. Obtain a renal biopsy to establish a diagnosis and prognosis (See section on Renal Biopsy). Administer gastroprotectant drugs and antiemetics to control nausea and vomiting. If possible, avoid the use of nephrotoxic drugs and general anesthesia. Initiate nutritional support in the form of an enteral feeding tube or parenteral nutrition as early as possible.

Once the patient enters the recovery phase, diuresis may occur that can lead to dehydration and electrolyte imbalances (hyponatremia, hypokalemia). Dehydration and electrolyte imbalances can be treated with parenteral fluid and electrolyte supplementation.

POSTRENAL AZOTEMIA

Postrenal azotemia is primarily caused by urethral obstruction or leakage from the urinary tract into the abdomen (uroabdomen). Complete urinary tract obstruction and uroabdomen are both ultimately fatal within 3 to 5 days if left untreated. In dogs, the most common causes of urethral obstruction are urinary (urethral) calculi or tumors of the urinary bladder or urethra. In male cats, feline urologic syndrome (FUS) is the most common cause of urethral obstruction, although there has been an increased incidence of urethral calculi observed in recent years. A ruptured urinary bladder is the most common cause of uroabdomen and is usually secondary to blunt trauma.

URINARY TRACT OBSTRUCTION

Clinical signs of urinary tract obstruction include dysuria, hematuria, inability to urinate or initiate an adequate stream of urine, and a distended painful urinary bladder. Late in the course of obstructive disease, clinical signs referable to uremia and azotemia (vomiting, oral ulcers, hematemesis, dehydration, lethargy, and anorexia) occur.

The initial goal of treatment of urinary tract obstruction is to relieve the obstruction. In male dogs, a lubricated catheter can be inserted past the area of obstruction with the animal under heavy sedation or general anesthesia (see section on Urohydropulsion). Depending on the chronicity of the obstruction, serum electrolytes should be measured; an ECG should be obtained before administering any anesthetic drugs, because of the cardiotoxic effects of hyperkalemia (see section on Atrial Standstill). Correct fluid, electrolyte, and acid-base abnormalities. If a urinary catheter cannot be placed, perform cystocentesis only as a last resort, because of the risk of urinary bladder rupture.

Definitive treatment includes identification and treatment of the underlying cause (tumor versus urinary calculi). In most cases, surgical intervention is necessary. If an unresectable tumor is present, a low-profile permanent cystostomy tube can be placed, if the owner desires. Administration of piroxicam (Feldene, 0.3 mg/kg PO q24-48h) with or without chemotherapy may shrink the tumor mass and delay the progression of clinical signs.

FELINE LOWER URINARY TRACT DISEASE

A complete discussion of this disorder is beyond the scope of this text (See Additional Reading for other sources of information). Feline lower urinary tract disease can cause urethral obstruction, particularly in male cats. Clinical signs include stranguria, dribbling of small amounts of urine, lethargy, inappetence, and vomiting. Often, owners call with the primary complaint of constipation, because the cat is making frequent trips to the litterbox and straining. Cases with a duration of obstruction < 36 hours are considered uncomplicated; those with a duration > 36 hours are complicated.

Treatment of urethral obstruction includes stabilizing and normalizing the patient's electrolyte status, induction of sedation or general anesthesia, and relieving the obstruction.

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Obtain blood samples for analysis of electrolyte abnormalities. Treat hyperkalemia ($K^+ > 6.0$ mEq/L) with sodium bicarbonate (0.25-1.0 mEq/kg IV), regular insulin (0.25 unit/kg IV) plus dextrose (1 g/unit of insulin IV), followed by 2.5% dextrose IV CRI to prevent hypoglycemia; or calcium gluconate (0.2 mL/kg 10% IV slowly). Administer non-potassium-containing intravenous fluids in 0.9% saline solution. Obtain an ECG to detect atrial standstill (see section on Atrial Standstill).

In some cases, a urethral plug is visible at the tip of the penis. The urethral plug can sometimes be manually extracted or massaged from the penis, and the obstruction temporarily relieved. In such cases, it is still necessary to pass a urethral catheter to flush sediment from the urethra and urinary bladder. Unless a patient is obtunded, administer an anesthetic such as ketamine, atropine, or propofol (4-7 mg/kg IV) with diazepam IV for patient comfort and muscle relaxation.

Once the patient is under anesthesia or heavily sedated, urinary catheterization should be performed. In some cases, it will be difficult to advance the catheter. Lubricate a closed-ended Tomcat catheter and pass the tip into the distal urethra. Fill a 12-mL syringe with sterile saline and sterile lubricant and connect the syringe to the hub of the catheter. Pulse the fluid into the catheter as you gently move the catheter tip back and forth against the urethral obstruction. When the catheter has been passed into the urinary bladder, obtain a urine sample for urinalysis. Drain the bladder and flush with sterile saline solution until the urine efflux appears clear. Remove the Tomcat catheter and insert a 3-5 Fr red rubber tube or Argyle infant feeding catheter into the urethra for urine collection and quantitation. Secure the urinary catheter to prepuce with a butterfly strip of 1-inch adhesive tape secured around the catheter and then sutured to either side of the prepuce. The catheter should be connected to a closed urinary collection system for cleanliness and to reduce the risk of ascending bacterial infection. An Elizabethan collar should be placed at all times to prevent the patient from damaging or removing the catheter.

When the urethral obstruction has been relieved and the catheter placed, continue intravenous fluid diuresis to alleviate postrenal azotemia. Monitor the urine for bacteria and other sediment. In some cases, postobstructive diuresis can be severe. Carefully monitor fluid ins and outs, along with body weight, to maintain adequate hydration and perfusion. Remove the urinary catheter can be removed after 24 to 48 hours. Palpate the bladder frequently to make sure that the patient is voiding normally and to detect the recurrence of obstruction.

In patients with severe penile or urethral trauma or edema, administer a short-acting steroid (dexamethasone sodium phosphate, 0.25 mg/kg IV, IM, SQ). At the time of initial diagnosis and again at the time of discharge, the clients need to be instructed about the long-term management of feline lower urinary tract disease at home, and informed of the risks and consequences of recurrence.

UROABDOMEN

Uroabdomen can occur from trauma or leakage from the kidneys, ureter, or urinary bladder. Clinical signs of uroabdomen (azotemia, uremia, hyperkalemia) can also occur secondary to third spacing of urine and leakage into muscular tissue from a ruptured urethra. In most cases, urinary bladder trauma and rupture are secondary to blunt trauma. Abdominocentesis should be performed in any animal with suspected blunt abdominal trauma, and any fluid obtained should be analyzed for creatinine or potassium and compared with the patient's serum levels. An abdominal effusion that has a low packed cell volume and a potassium or creatinine level greater than that of the patient's serum is consistent with the diagnosis of uroabdomen.

Uroabdomen is not a surgical emergency. However, medical management consists of placement of a temporary abdominal drainage catheter into the abdomen, to facilitate removal of urine from the peritoneal cavity. To place the catheter, position the patient in dorsal or lateral recumbency, shave the ventral abdomen, as for any exploratory laparotomy. Aseptically scrub the clipped area, and instill a local anesthetic (lidocaine, 1-2 mg/kg) caudal and to the right of the umbilicus, through the skin, subcutaneous tissues, and rectus

abdominis muscles, inserting the lidocaine as you pull the needle out, thus creating an anesthetized tunnel. Aseptically scrub the area again and drape with sterile field towels; then make a small stab incision through the skin. Bluntly dissect through the subcutaneous tissue to the level of the external rectus abdominis. Pick up the muscle with a thumb forceps, and make a small stab incision into the abdominal cavity. Cut multiple holes in the side of a 14-16 Fr red rubber tube or thoracic drainage catheter, using care not to make the cut wider than 50% of the circumference of the tube. Insert the catheter into the abdominal cavity in a dorsal caudal direction. Make sure that all incisions within the abdomen. Secure the tube by placing a pursestring suture around the tube entrance site in the abdominal musculature with absorbable suture material. Close the dead space in the subcutaneous tissues with absorbable suture. Close the skin around the tube with another purse-string suture secured using a finger-trap technique. Connect the tube to a closed urinary collection system and bandage the catheter to the abdomen. The tube can remain in place until the patient's cardiorespiratory status is stabilized enough to allow anesthesia and definitive repair of the urinary tract defect.

Additional Reading

- Forrester SD, McMillan NS, Ward DL: Retrospective evaluation of acute renal failure in dogs. *J Vet Intern Med* 16:354, 2002.
- Gannon KM, Moses L: Uroabdomen in dogs and cats. *Comp Cont Educ Pract Vet* 248: 604-612, 2002.
- Geor RJ: Drug-induced nephrotoxicity: recognition and prevention. *Comp Cont Educ Pract Vet* 22(9):876-888, 2000.
- Labato MA: Peritoneal dialysis in emergency and critical care. *Clin Tech Small Anim Pract*. 15:126-135, 2000
- Langston CE: Acute renal failure caused by lily ingestion in six cats. *J Am Vet Med Assoc* 220(1):49-52, 2002.
- Lees GE: Early diagnosis of renal disease and renal failure. *Vet Clin North Amer Sm Anim* 34:867-885, 2004.
- Mazzaferro EM, Eubig PE, Hackett TB, et al: Acute renal failure in four dogs after raisin or grape ingestion (1999-2002). *J Vet Emerg Crit Care* 14(3):203-212, 2004.
- Osborne CA, Kruger JM, Lulich JP, et al: Disorders of the feline lower urinary tract. In Osborne CA, Finco DR, editors: *Canine and feline nephrology and urology*, Baltimore, 1995, Williams and Wilkins.
- Rieser TM: Urinary tract emergencies. *Vet Clin North Am Small Anim Pract* 35:359-373, 2005.
- Salinardi BJ, Marks SL, Davidson JR, Senior DF: The use of a low-profile cystostomy tube to relieve urethral obstruction in a dog. *J Am Anim Hosp Assoc* 39(4):403-405, 2003.
- Stokes JE, Forrester SD: New and unusual cases of acute renal failure in dogs and cats. *Vet Clin North Am Small Anim Pract* 34:909-922, 2004.
- Vaden SL: Renal biopsy: methods and interpretation. *Vet Clin North Am Small Anim Pract* 34:887-908, 2004.
- Westropp JL, Buffington CAT: Feline idiopathic cystitis: current understanding of pathophysiology and management. *Vet Clin North Am Small Anim Pract* 34:1043-1055, 2004.